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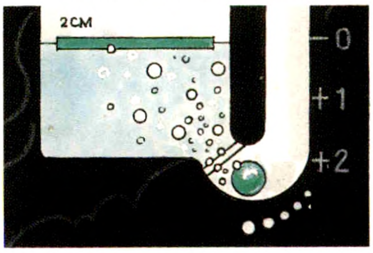

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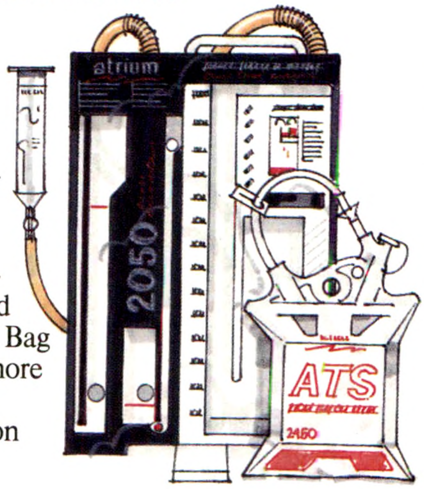

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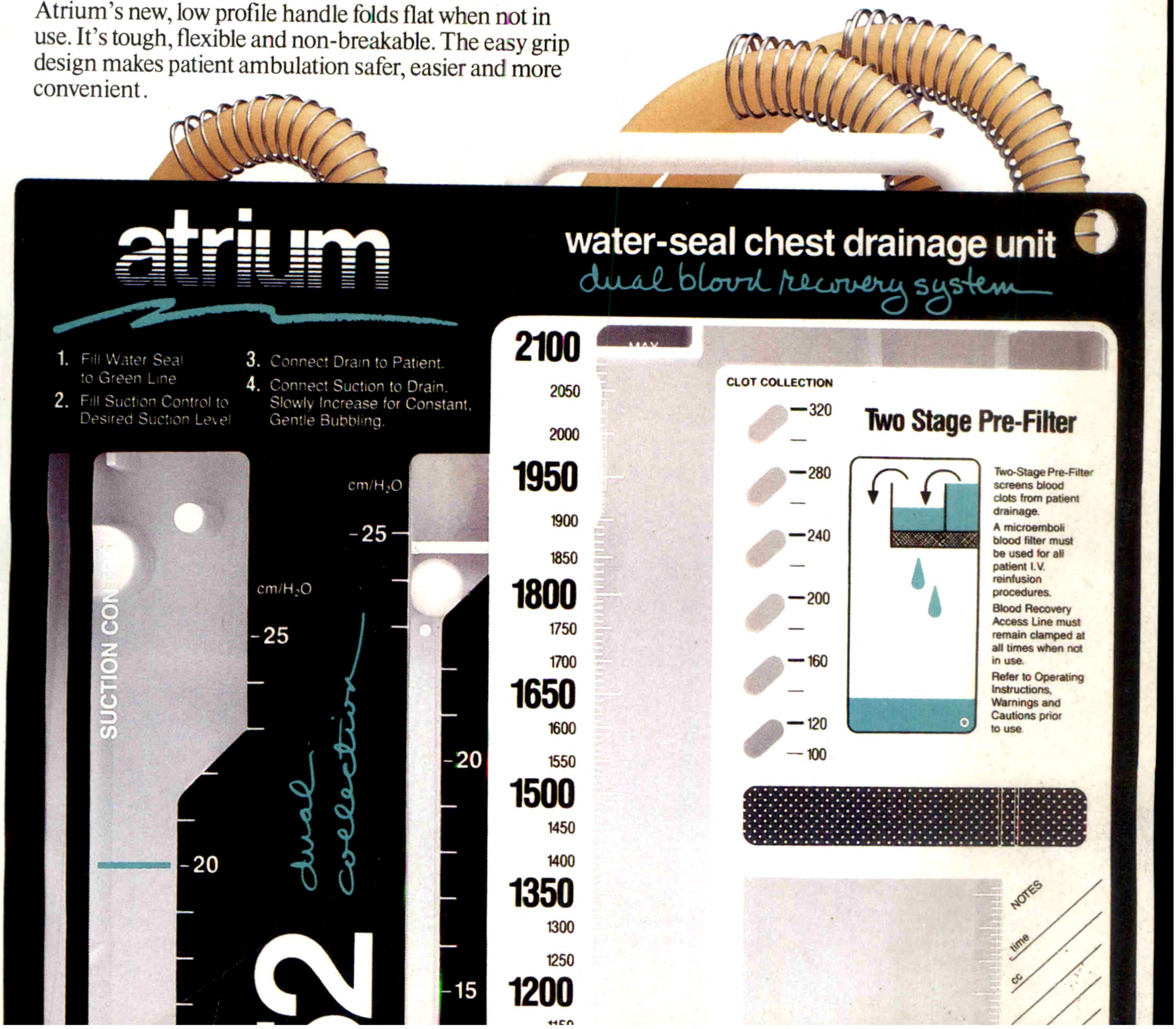

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1. Heinemann F, et al: Clinical comparison of available 'low threshold' leads. European Society of Cardiology, Congress, Vienna, 1988.
2. Steinhaus DM, Foley LM: Atrial sensing: A continuing problem? (abstract) *PACE* 1988;11(4):533.
3. IS-1 is the designation of the standard pacing connector developed by the International Committee on Cardiovascular Implants for Surgery (International Standard Organization ISO TC150/SC).

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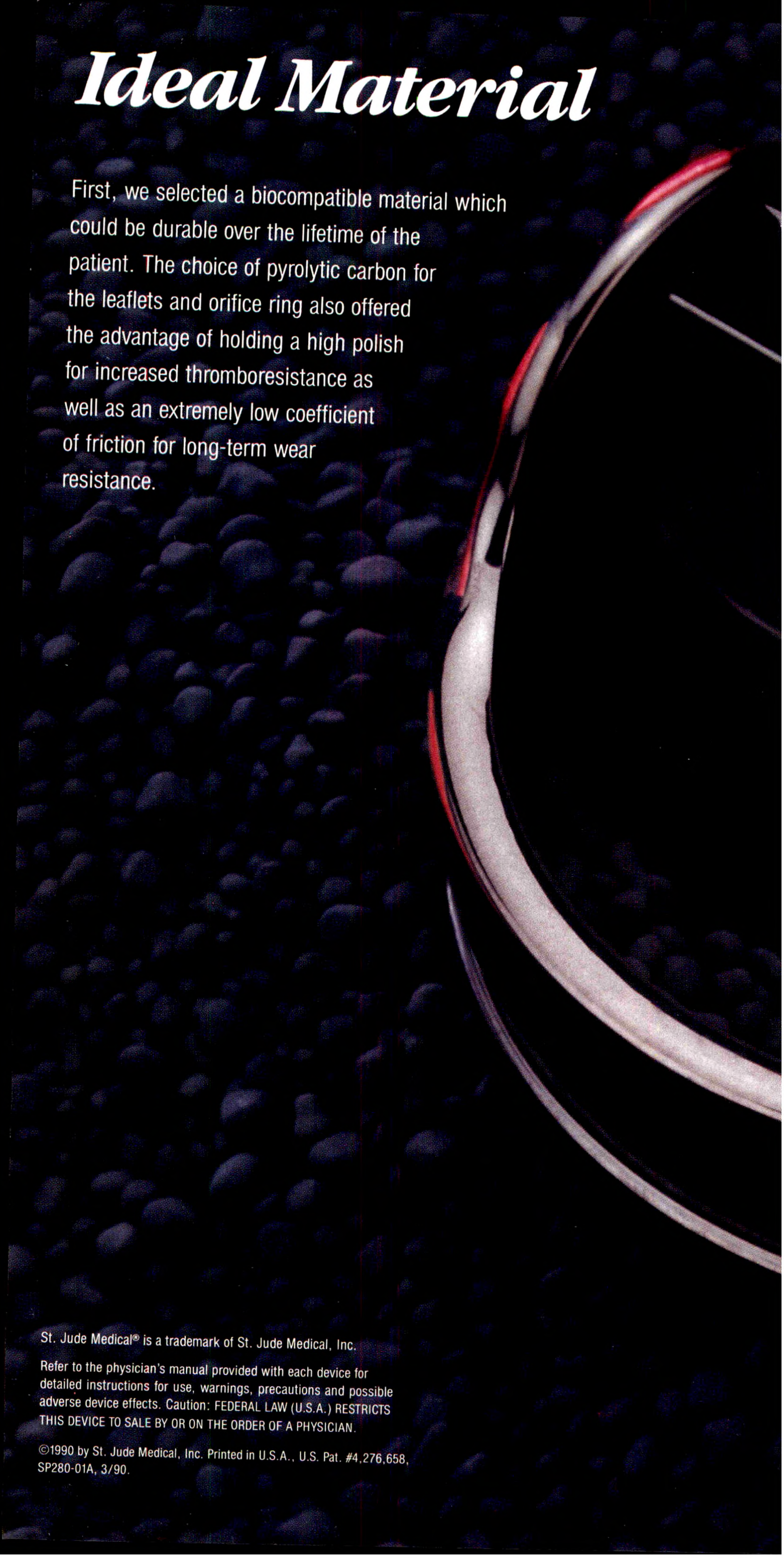

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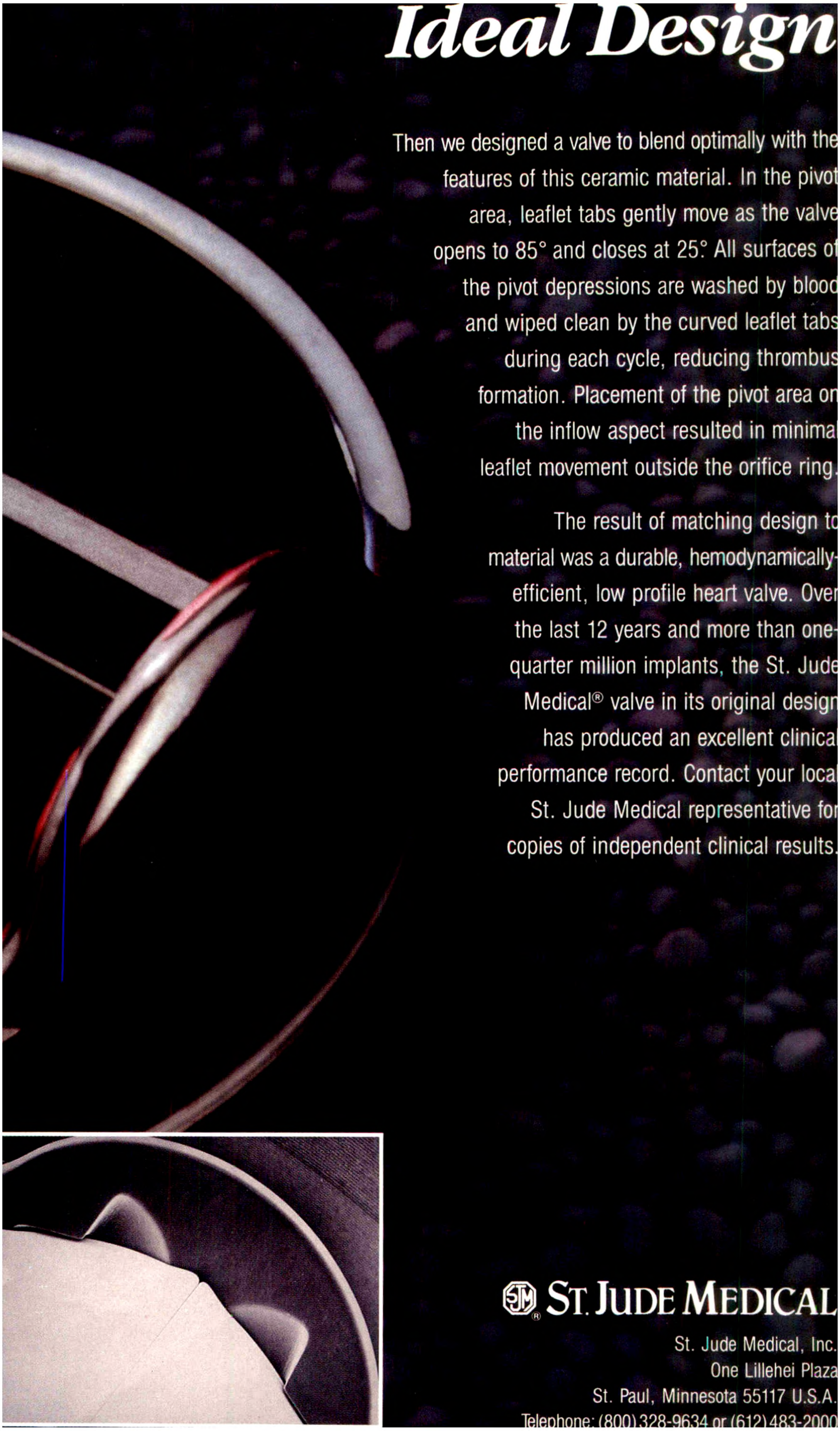
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Lung and Heart-Lung Transplantation
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- ☐ Arrange manuscript as follows: (1) title page, (2) abstract, (3) text, (4) references, (5) tables, and (6) legends. Number pages consecutively, beginning with the title page as page 1 and ending with the legend page.
- ☐ A manuscript for an *original article* ordinarily should not exceed 26 double-spaced typewritten pages, and should be proportionally shorter the more illustrations and tables that are included.
- ☐ The *text* for case reports should be *no more* than 4 double-spaced typewritten pages. If tables or illustrations are included, the text must be reduced. The text for a "how to do it" article should be even briefer, but with detailed illustrative material. Case reports and "how to do it" articles that exceed the allowable length will not be considered for publication.
- ☐ Referencing should be selective and pertain directly to the work being reported. Except in collective and current reviews, comprehensive listings serve no useful purpose and utilize valuable space. As a general guide, original articles should have no more than 20 references; case reports, 8 references; and "how to do it" articles, 5 references.

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- ☐ Give the paper as short a title as possible. Also submit a short title of 3 or 4 words to be used as a running head. Include only the names of the authors directly affiliated with the work, stating for each the highest academic degree. Include the name and location of no more than two institutional affiliations.
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Chapter in Book

12. Katz PO. Achalasia. In: Castell DO, Richter JE, Dalton CB, eds. Esophageal motility testing. New York: Elsevier Science Publishing Company, 1987:107.

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> **WARNING**
>
> It is recommended that Blenoxane be administered under the supervision of a qualified physician experienced in the use of cancer chemotherapeutic agents. Appropriate management of therapy and complications is possible only when adequate diagnostic and treatment facilities are readily available.
>
> Pulmonary fibrosis is the most severe toxicity associated with Blenoxane. The most frequent presentation is pneumonitis occasionally progressing to pulmonary fibrosis. Its occurrence is higher in elderly patients and in those receiving greater than 400 units total dose, but pulmonary toxicity has been observed in young patients and those treated with low doses.
>
> A severe idiosyncratic reaction consisting of hypotension, mental confusion, fever, chills, and wheezing has been reported in approximately 1% of lymphoma patients treated with Blenoxane.

DESCRIPTION

Blenoxane (sterile bleomycin sulfate, USP) is a mixture of cytotoxic glycopeptide antibiotics isolated from a strain of **Streptomyces verticillus.** It is freely soluble in water.

Note: A unit of bleomycin is equal to the formerly used milligram activity. The term milligram activity is a misnomer and was changed to units to be more precise.

ACTION

Although the exact mechanism of action of Blenoxane is unknown, available evidence would seem to indicate that the main mode of action is the inhibition of DNA synthesis with some evidence of lesser inhibition of RNA and protein synthesis.

In mice, high concentrations of Blenoxane are found in the skin, lungs, kidneys, peritoneum, and lymphatics. Tumor cells of the skin and lungs have been found to have high concentrations of Blenoxane in contrast to the low concentrations found in hematopoietic tissue. The low concentrations of Blenoxane found in bone marrow may be related to high levels of Blenoxane degradative enzymes found in that tissue.

In patients with a creatinine clearance of >35 mL per minute, the serum or plasma terminal elimination half-life of bleomycin is approximately 115 minutes. In patients with a creatinine clearance of <35 mL per minute, the plasma or serum terminal elimination half-life increases exponentially as the creatinine clearance decreases. In humans, 60% to 70% of an administered dose is recovered in the urine as active bleomycin.

INDICATIONS

Blenoxane should be considered a palliative treatment. It has been shown to be useful in the management of the following neoplasms either as a single agent or in proven combinations with other approved chemotherapeutic agents:

Squamous Cell Carcinoma—Head and neck (including mouth, tongue, tonsil, nasopharynx, oropharynx, sinus, palate, lip, buccal mucosa, gingiva, epiglottis, larynx), skin, penis, cervix, and vulva. The response to Blenoxane is poorer in patients with head and neck cancer previously irradiated.

Lymphomas—Hodgkin's, reticulum cell sarcoma, lymphosarcoma.

Testicular Carcinoma—Embryonal cell, choriocarcinoma, and teratocarcinoma.

CONTRAINDICATIONS

Blenoxane is contraindicated in patients who have demonstrated a hypersensitive or an idiosyncratic reaction to it.

WARNINGS

Patients receiving Blenoxane must be observed carefully and frequently during and after therapy. It should be used with extreme caution in patients with significant impairment of renal function or compromised pulmonary function.

Pulmonary toxicities occur in 10% of treated patients. In approximately 1%, the nonspecific pneumonitis induced by Blenoxane progresses to pulmonary fibrosis, and death. Although this is age and dose related, the toxicity is unpredictable. Frequent roentgenograms are recommended.

Idiosyncratic reactions similar to anaphylaxis have been reported in 1% of lymphoma patients treated with Blenoxane. Since these usually occur after the first or second dose, careful monitoring is essential after these doses.

Renal or hepatic toxicity, beginning as a deterioration in renal or liver function tests, have been reported infrequently. These toxicities may occur, however, at any time after initiation of therapy.

Usage in Pregnancy: Safe use of Blenoxane in pregnant women has not been established.

ADVERSE REACTIONS

Pulmonary—This is potentially the most serious side effect, occurring in approximately 10% of treated patients. The most frequent presentation is pneumonitis occasionally progressing to pulmonary fibrosis. Approximately 1% of patients treated have died of pulmonary fibrosis. Pulmonary toxicity is both dose and age related, being more common in patients over 70 years of age and in those receiving over 400 units total dose. This toxicity, however, is unpredictable and has been seen occasionally in young patients receiving low doses.

Because of lack of specificity of the clinical syndrome, the identification of patients with pulmonary toxicity due to Blenoxane has been extremely difficult. The earliest symptom associated with Blenoxane pulmonary toxicity is dyspnea. The earliest sign is fine rales.

Radiographically, Blenoxane-induced pneumonitis produces nonspecific patchy opacities, usually of the lower lung fields. The most common changes in pulmonary function tests are a decrease in total lung volume and a decrease in vital capacity. However, these changes are not predictive of the development of pulmonary fibrosis.

The microscopic tissue changes due to Blenoxane toxicity include bronchiolar squamous metaplasia, reactive macrophages, atypical alveolar epithelial cells, fibrinous edema, and interstitial fibrosis. The acute stage may involve capillary changes and subsequent fibrinous exudation into alveoli producing a change similar to hyaline membrane formation and progressing to a diffuse interstitial fibrosis resembling the Hamman-Rich syndrome. These microscopic findings are nonspecific, eg, similar changes are seen in radiation pneumonitis, pneumocystic pneumonitis.

To monitor the onset of pulmonary toxicity, roentgenograms of the chest should be taken every 1 to 2 weeks. If pulmonary changes are noted, treatment should be discontinued until it can be determined if they are drug related. Recent studies have suggested that sequential measurement of the pulmonary diffusion capacity for carbon monoxide (DL_{CO}) during treatment with Blenoxane may be an indicator of subclinical pulmonary toxicity. It is recommended that the DL_{CO} be monitored monthly if it is to be employed to detect pulmonary toxicities, and thus the drug should be discontinued when the DL_{CO} falls below 30% to 35% of the pretreatment value.

Because of bleomycin's sensitization of lung tissue, patients who have received bleomycin are at greater risk of developing pulmonary toxicity when oxygen is administered in surgery. While long exposure to very high oxygen concentrations is a known cause of lung damage, after bleomycin administration, lung damage can occur at lower concentrations than usually would be considered safe. Suggested preventive measures are:

(1) Maintain FI O_2 at concentrations approximating that of room air (25%) during surgery and the postoperative period.
(2) Monitor carefully fluid replacement, focusing more on colloid administration rather than crystalloid.

Sudden onset of an acute chest pain syndrome suggestive of pleuropericarditis has been rarely reported during Blenoxane infusions. Although each patient must be individually evaluated, further courses of Blenoxane do not appear to be contraindicated.

Idiosyncratic Reactions—In approximately 1% of the lymphoma patients treated with Blenoxane, an idiosyncratic reaction, similar to anaphylaxis clinically, has been reported. The reaction may be immediate or delayed for several hours, and usually occurs after the first or second dose. It consists of hypotension, mental confusion, fever, chills, and wheezing. Treatment is symptomatic including volume expansion, pressor agents, antihistamines, and corticosteroids.

Integument and Mucous Membranes—These are the most frequent side effects being reported in approximately 50% of treated patients. These consist of erythema, rash, striae, vesiculation, hyperpigmentation, and tenderness of the skin. Hyperkeratosis, nail changes, alopecia, pruritus, and stomatitis have also been reported. It was necessary to discontinue Blenoxane therapy in 2% of treated patients because of these toxicities.

Skin toxicity is a relatively late manifestation usually developing in the 2nd and 3rd week of treatment after 150 to 200 units of Blenoxane have been administered and appears to be related to the cumulative dose.

Other—Vascular toxicities coincident with the use of Blenoxane in combination with other antineoplastic agents have been reported rarely. The events are clinically heterogeneous and may include myocardial infarction, cerebrovascular accident, thrombotic microangiopathy (HUS) or cerebral arteritis. Various mechanisms have been proposed for these vascular complications. There are also reports of Raynaud's phenomenon occurring in patients treated with Blenoxane in combination with vinblastine with or without cisplatin or, in a few cases, with Blenoxane as a single agent. It is currently unknown if the cause of Raynaud's phenomenon in these cases is the disease, underlying vascular compromise, Blenoxane, vinblastine, hypomagnesemia, or a combination of any of these factors.

Fever, chills, and vomiting were frequently reported side effects. Anorexia and weight loss are common and may persist long after termination of this medication. Pain at tumor site, phlebitis, and other local reactions were reported infrequently.

DOSAGE

Because of the possibility of an anaphylactoid reaction, lymphoma patients should be treated with 2 units or less for the first two doses. If no acute reaction occurs, then the regular dosage schedule may be followed.

The following dose schedule is recommended: **Squamous cell carcinoma, lymphosarcoma, reticulum cell sarcoma, testicular carcinoma**—0.25 to 0.50 units/kg (10 to 20 units/m^2) given intravenously, intramuscularly, or subcutaneously weekly or twice weekly.

Hodgkin's Disease—0.25 to 0.50 units/kg (10 to 20 units/m^2) given intravenously, intramuscularly, or subcutaneously weekly or twice weekly. After a 50% response, a maintenance dose of 1 unit daily or 5 units weekly intravenously or intramuscularly should be given.

Pulmonary toxicity of Blenoxane appears to be dose related with a striking increase when the total dose is over 400 units. Total doses over 400 units should be given with great caution.

Note: When Blenoxane is used in combination with other antineoplastic agents, pulmonary toxicities may occur at lower doses.

Improvement of Hodgkin's Disease and testicular tumors is prompt and noted within 2 weeks. If no improvement is seen by this time, improvement is unlikely. Squamous cell cancers respond more slowly, sometimes requiring as long as 3 weeks before any improvement is noted.

ADMINISTRATION

Blenoxane may be given by the intramuscular, intravenous, or subcutaneous routes.

Intramuscular or Subcutaneous—Dissolve the contents of a Blenoxane vial in 1 to 5 mL of Sterile Water for Injection, USP, Sodium Chloride for Injection, USP, 5% Dextrose Injection, USP, or Bacteriostatic Water for Injection, USP.

Intravenous—Dissolve the contents of the vial in 5 mL or more of a solution suitable for injection, eg, physiologic saline or glucose, and administer slowly over a period of 10 minutes.

STABILITY

The sterile powder is stable under refrigeration (2°-8°C) and should not be used after the expiration date is reached.

Blenoxane is stable for 24 hours at room temperature in Sodium Chloride or 5% Dextrose Solution.

Blenoxane is stable for 24 hours in 5% Dextrose containing heparin 100 units per mL or 1000 units per mL.

Procedures for proper handling and disposal of anticancer drugs should be considered. Several guidelines on this subject have been published.[1-7] There is no general agreement that all of the procedures recommended in the guidelines are necessary or appropriate.

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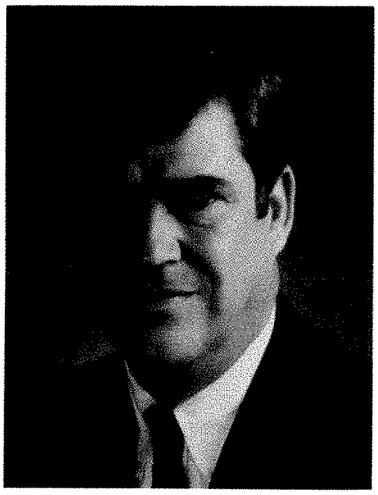

I thank you, the membership of the Southern Thoracic Surgical Association for asking me to be your president this year. It is a singular honor in my life, and I truly appreciate the opportunity to serve you. I thank Mark Braimbridge for accepting the Special Speaker task and for coming such a long distance with his wife, Elizabeth. I wish to give special thanks to my wife, Barbara, for all the support she has provided. She has had remarkable sustained enthusiasm, sharing, and tolerance for my work. She has been extraordinarily patient waiting for things to get done at home because of all the days and weekends I have been away—at work or at play. My last thanks are to our progeny: Warren, Paul, John, Karen, and Todd, not only for their presence on this earth but because they have grown up to become decent, thoughtful, caring, and loving people.

My thoughts for this address ranged across a broad spectrum as, I am sure, did those of previous presidents. We all face many current pressing problems that need to be spoken about: malpractice and liability, the Health Care Financing Administration and Medicare payments, quality assurance and the hospital Gestapo, the cost-benefit ratio and Diagnosis-Related Groups, and many more. I could have railed against the current doctor-bashing by the press, where surgeons are a technical commodity and are characterized by greed. These topics were so discouraging that I thought I would rather talk about something far more interesting; namely, us.

Initially, I titled this address "The Pursuit of Excellence." The word *pursuit* seemed appropriate because it is an active verb meaning the act of chasing, striving; an act regularly engaged in; an avocation or a hobby. And the word *excellence* seemed to apply to us; the Latin base means "to rise high." Others suggested that the title should be more specific; hence the title "Who, Hobbies, and Heroes."

My purpose in talking about us is threefold. First, to describe with limited data a picture of who we are. How many are we? What is happening to us? What have we accomplished? What volume of services do we provide? What price do we pay? A second purpose is to suggest that high-performance, high-stress, no-margin-for-error professionals must have an outside interest, a hobby, to survive and to thrive. Finally, I will suggest to you that there is a need for heroes in our lives; an image to recall to point the way and encourage us in times of trouble, and to share in our triumphs.

Who Are We?

To help answer that question I have collected some data from a variety of sources. First, how many are we? The total number of board certificates issued to date is 4,900 (practicing, 4,421; deceased, 172; and retired, 307). For the last several years, about 134 candidates have received their certificates annually (personal communication, American Board of Thoracic Surgery and American Board of Medical Specialties, Evanston, IL). Of all the surgical specialties, cardiothoracic surgery is the eighth lowest in number of certificates issued per year with only neurological surgery and colorectal surgery being less at 118 and 42, respectively [1]. The number of cardiothoracic residents in training per year has been constant for the past 5 years. The 1987 manpower report estimated the number of practicing cardiothoracic surgeons at 3,795 [2].

How Old Are We?

It appears that we are an aging population. The average age in 1980 and 1985 and estimated in 1990 show an increase from 48 to 50 to 52 years [2]. The number of those who are over 64 years of age has also increased from 47 in 1976 to 154 in 1985. I estimate that in 1990, 217 surgeons will reach the age of 65 years.

The data from the American College of Surgeons, supplied by Dr Ebert's office, showed that the retirement age of all surgeons is decreasing (Fig 1). The active age bracket is between the ages of 35 and 54 years. Sixty-one percent of all general thoracic procedures are done by this age group. Impressively, 85% of all cardiac procedures and 75% of all cardiothoracic operations are performed by surgeons 35 to 54 years of age. Can we characterize those older than 50 years? The manpower report demonstrated

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Presented at the Thirty-sixth Annual Meeting of the Southern Thoracic Surgical Association, Scottsdale, AZ, Nov 9–11, 1989.

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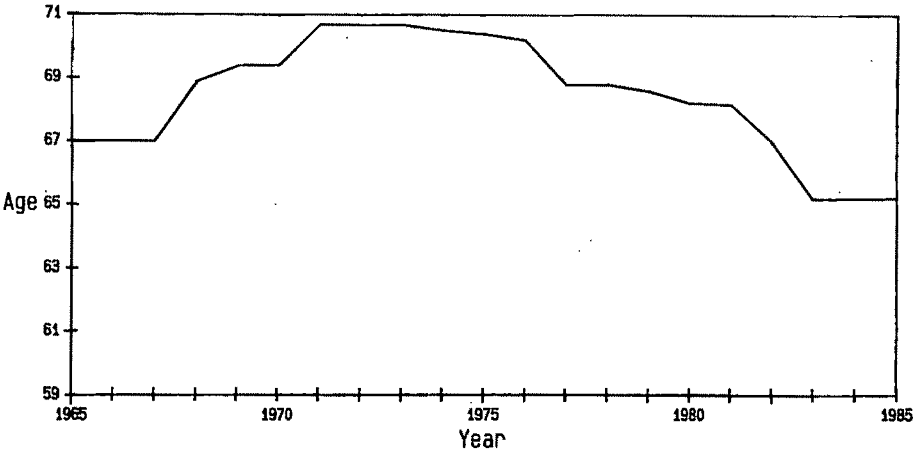

Fig 1. Retirement age (3-year weighted average) of Fellows of the American College of Surgeons by year. (Graphic kindly supplied by Paul A. Ebert, MD, Director, American College of Surgeons, Chicago, IL.)

that those older than 50 years performed more general thoracic operations, 54 versus 37 per year, and concomitantly fewer cardiac operations (120 versus 56). Striking was the statement that 50% of surgeons older than 50 years did not perform cardiac operations at all.

Thus, it appears that those who initiated cardiac operations and are now aging have shown a self-selection tendency. Perhaps this is analogous to playing strongside line backer for the National Football League. There is a finite period that one can do cardiac surgery and do it well.

Another indicator of the aging population is the age of the members of The Society of Thoracic Surgeons. Shown in Figure 2 are the percentages by age of the members for whom The Society has birth data. Fifty-six percent are older than 50 years, and 34% are older than 55 years. Only 22% are in the 36- to 45-year age group.

Other demographics show that over the past decade fewer surgeons are going into solo practice and that most (64%) prefer to practice in only one or two hospitals [2]. The highest concentration of cardiothoracic surgeons is on the West Coast, and the lowest is in the Southeast central area; that area covered by the Southern Thoracic Surgical Association [2]. The number of surgeons practicing in communities with more than 100,000 residents has been steadily going up, and the projection is that approximately 90% will be in such communities in 1990 [2].

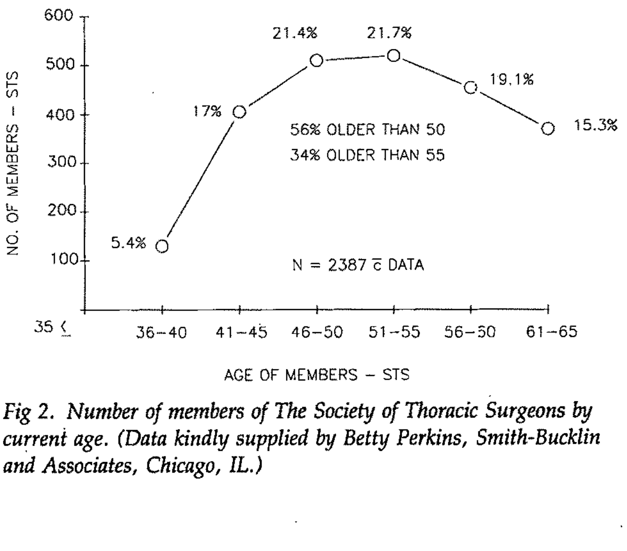

Fig 2. Number of members of The Society of Thoracic Surgeons by current age. (Data kindly supplied by Betty Perkins, Smith-Bucklin and Associates, Chicago, IL.)

Having given you some of the demographics, what do we do? The numbers are absolutely phenomenal. The 1985 survey showed that the 2,347 respondents provided 447,555 operations in 1985. Indeed, the total number of operations is increasing by more than 5% every 5 years. Shown in Figure 3 are the trends of increasing procedures over the past decade.

Easier to digest are the data in Table 1 for the number and type of procedures and the number of surgeons performing the operations in 1985. This amount of work is truly remarkable. Moreover, when one takes the data from the respondents and uses the estimate of 3,795 active surgeons, the specialty of cardiothoracic surgery provided 663,965 major operations, with 467,733 being cardiovascular and the balance being general thoracic. These data are very similar to those supplied by the National Center for Health Care Statistics as an estimate for procedures in that year [2].

What About You, the Individual Surgeon?

In 1985, each of you carried out an average of 258 procedures, the majority of which were cardiac in nature. There were 140 cardiac, 48 general thoracic, 47 peripheral vascular, and 23 pacemaker procedures per individual practicing cardiothoracic surgeon [2].

Many surgeons are engaged in other activities. As of September 1, 1989, we are currently training approximately 295 residents in 95 approved US programs. In addition to teaching and training, we have been busy in publishing. I believe that my estimate of more than 600 research and clinical papers by US surgeons for 1988 is probably low.

Let me speak for a moment about the research area as a part of the Who. A relative minority of cardiothoracic surgeons have wandered over or have become enamored with research. Associations, societies, foundations, and universities are becoming acutely aware of the need for clinically well-trained cardiothoracic surgeons who also are well trained and motivated to do clinical and laboratory research.

In his presidential address before the Association of

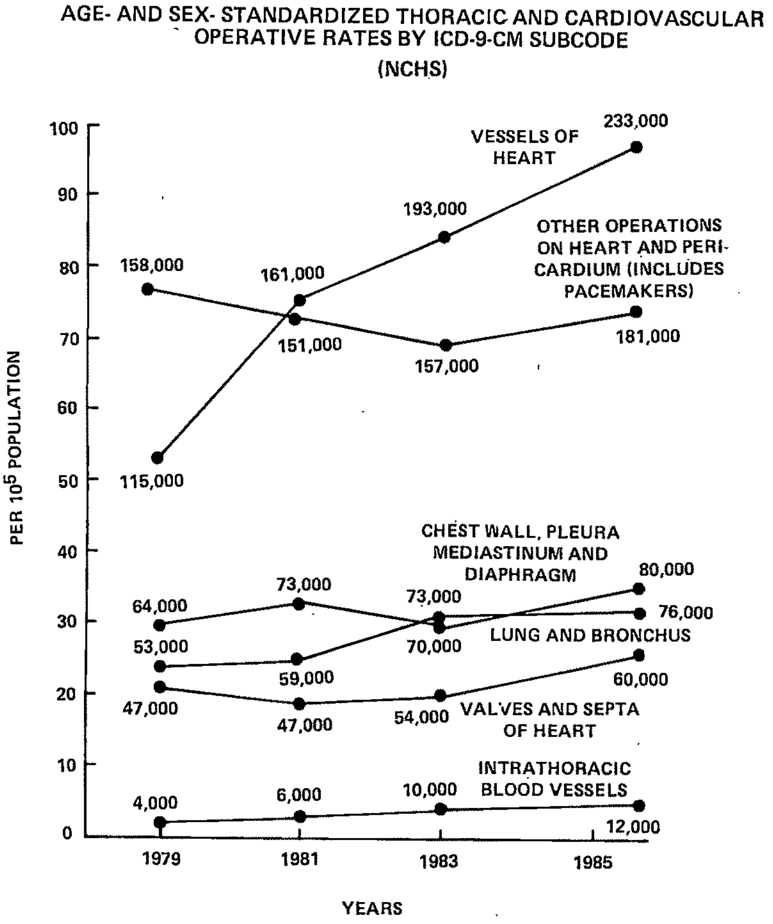

Fig 3. Number of cardiovascular procedures performed by certified cardiothoracic surgeons by year. (Graphic kindly supplied by Floyd D. Loop, MD, Cleveland, OH, updated from original statistics supplied by Ira M. Rutkow, MD, Newark, NJ.)

Academic Surgeons, Judah Folkman [3] characterized those clinical surgeons who did research as a paradox. He listed four obstacles for the young surgeon/researcher: carry your weight, reverse discrimination, the spouse-activator factor, and the surgical personality.

The first obstacle (carry your weight) suggests that you must develop a practice to meet all your expenses and not be a financial burden on the rest of the group. Hence, the majority of the young surgeon's energy should be channeled into the clinical arena and not into the classroom or laboratory.

The second obstacle is reverse discrimination in which, had you gone to the laboratory, you would have become known as the rat or the dog doctor by your capricious competitors.

Table 1. 1985 Activity of Certified Cardiothoracic Surgeons[a]

Type	No. of Procedures	No. of Surgeons
General thoracic	107,884	2,236
Cardiac	221,036	1,579
Transplant	1,789	
Thoracic aneurysms	5,453	
Arrhythmic	1,447	
Pacemakers	36,113	1,586
Peripheral vascular	72,522	1,560

[a] Data rearranged from reference 3.

The third item is the spouse-activator factor. This is the domestic confrontation that occurs toward the end of a long residency. The spouse says, "The furniture is worn out, the car is costing too much in repairs, and the kids don't have decent clothes. You need to get out there and get the best paying job you can. You can't accept the measly salary you were offered as an assistant professor so you could do research."

The surgical personality has two characteristics that are diametrically opposed to those required in research according to Dr Folkman. First, the surgeon has a sense of time urgency, which is "an abiding fear that someone will waste his time." The second characteristic that makes research difficult for the surgeon is doubt. "He may often be wrong but he is never in doubt."

Dr Louis G. D'Alecy, a professor of surgery and physiology at the University of Michigan, has a chapter in a textbook entitled *Medical Education: A Surgical Perspective.* The telling title of his chapter is "Surgeon scientist, adaptive schizophrenia: a view from the other side" [4]. The point of the chapter, much like that of Folkman, is that the surgeon does not have a contemplative, temperate, or hesitant personality or practice. Surgeons are clear of purpose, definitive in judgment, decisive, and authoritative.

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In contrast, much of research is spent in discussion, speculation, reading, and analyzing. Research concerns are testable hypotheses, inconsistencies in data, and searches for new solutions. In research, asking a question may be more important than the answer. One has to doubt frequently and be willing to be wrong. Finally, in D'Alecy's view, enhancement of technical skills is but a fringe benefit for the trainee in surgical research.

Morrow, in his presidential address to the Society of Vascular Surgery in 1972 [5], had as his subject "What the cardiac surgeon should be." He noted that there was a role for the surgeon in research: "He is in an excellent position to bridge the applicability gap between the observations made by the basic scientist in the treatment of the patient with heart disease. It is in this area that the cardiac surgeon will contribute as an investigator."

According to M. H. Fisher, "Research has been called good business, a necessity, a gamble, a game. It is none of these. It's a state of mind."

I submit to you that the opportunity to have a professional life in the world of ideas, the joy of constant learning, the euphoria of discovery—no matter how small—can be deeply satisfying. Dr Richard Varco [6] expressed this feeling in his presidential address by saying, "to improve for man the future by the device of mind is to sip of heady wine."

Do We Pay a Price?

First, let us look at the amount of energy put out by the operating surgeon. Data obtained in England demonstrate that while operating, heart rates of surgeons performing abdominal procedures reached a maximum of 114 beats/min, nearly twice the sleeping heart rates. These heart rates were considerably higher for the amount of oxygen actually used than those when exercising on a bicycle to the same level of oxygen consumption [7]. The oxygen consumption while operating (16% of maximum) is slightly more than that which physiologists have found for a standing 45-year-old man (345 mL/min). The first assistant used one third less oxygen per minute than the surgeon. This oxygen consumption is a very small proportion of the maximal oxygen consumption, which is in the range of 3 to 4 L/min [8].

Albumin excretion has been measured in operating surgeons as an index of the level of sympathetic tone and possible reflection of the increases in heart rate and blood pressure. Before operation, a very small amount of albumin was excreted in 6 surgeons with normal renal function. During operation, the values increased nearly threefold. Normal excretion patterns were obtained when the surgeons worked in the Outpatient Clinic. This finding was age related and appeared to be a phenomenon associated with those in training. The highest excretion patterns were those in the young trainee, and the lowest were in those older than 40 years [9].

Other heart rate studies have compared surgeons with anesthesiologists during cardiothoracic operations. Cardiothoracic surgeons, at least in terms of heart rate, work at least 25% harder than anesthesiologists. In terms of the hours worked per day in England, this was also true (14.7 versus 10.5 hours). Mean heart rates by occupation have been compared. Three studies demonstrated increased heart rates for surgeons during operations, compared with those of anesthesiologists and managers, who had the lowest. Surgeons during operations, in terms of their heart rate, are working at about the rate of professional fisherman hauling nets on the open sea [10].

Other measures of stress besides heart rate and oxygen consumption have been reported in psychological studies that have been conducted particularly over the last decade. One study compared the stress and practice patterns in 50 lawyers and 50 physicians [11]. These data seem to support our common characterization of the lawyer. First, the lawyer was twice as tolerant to frustration as the doctor. Second, the lawyer's reaction to stress was most likely to be a decrease in activity, whereas the physician's response to stress was more likely to be an increase in activity. Lawyers had twice the incidence of substance abuse. The greatest frustrations were somewhat similar. The lawyer was most frustrated by the noncompliant client, and the doctor was similarly frustrated by the noncompliant patient. Lawyers were angered when they were not paid. Doctors, on the other hand, were angered when nurses refused to carry out their orders. Both groups were very frustrated by excessive paperwork. There were marked differences in compulsive features. More than 80% of doctors were characterized as highly compulsive with 20% being in the disorder range. Lawyers, in contrast, were rather noncompulsive people. Finally, the relaxation time taken for true vacations was twice as great for lawyers as it was for doctors. Do these characteristics of surgeons in practice patterns give rise to difficulty?

The stress syndrome apparently starts early at the completion of surgical training. Dr Johnnie Lamb [12], Chairman of the Department of Surgery at Travis Air Force Base, described the first-year attending surgeon's high-stress syndrome: a compulsive over-achiever, this person had the "Super Chief" attitude with inability to turn over daily responsibilities to the house staff. He lost his sense of humor and frequently lost his cool in the operating room when challenged by a difficult case. He was overly defensive, especially regarding complications and judgmental decisions. He was easily frustrated by organizational red tape. He truly had the "young-man-in-a-hurry syndrome."

These attitudes generally lasted 1 to 2 years until the staff surgeon became a bit more tolerant and was better able to interact with departmental personnel and assume a more supervisory role in training. This was concomitant with developing improved technical competence and increased confidence in his judgments. Job satisfaction became greater, and creativity increased proportionally as the stress decreased over time.

The older cardiothoracic surgeon thinks of himself as the Iron Man who is omnipotent, indestructible, in command, and self-sufficient. Psychological studies have demonstrated that surgeons, particularly cardiothoracic surgeons, have a high degree of need for control. We are demanding. We are perfectionists. We are highly compulsive, and yet we are all vulnerable. We are vulnerable to those who do not comply either with our wishes or with

Table 2. Percentage Distribution by Specialty of First One-Thousand Physicians Admitted to the Impaired Physician Program[a]

Specialty	Distribution of Physicians in Program (%)	AMA Distribution of US Physicians (%)	Incidence Ratio
General and family practice	25.7	12.4	2.0
Anesthesia	12.1	3.9	3.1
Emergency medicine	4.8	2.0	2.4
Thoracic surgery	1.2	0.4	3.0
Plastic surgery	1.5	0.7	2.1
Neurologic surgery	1.3	0.7	1.8

[a] Data taken from reference 15.

AMA = American Medical Association

our treatments. We may be more subject to disease, perhaps because of the high stress, but we do not know the total outcome yet because the specialty is very young. We have a problem with addiction and it is clear that surgeons in general have a serious inability to relax when they are out of the hospital.

One study has compared physicians with socioeconomic controls. There was a greater percentage of bad marriages or divorce on the part of physicians by 47%, a greater excessive use of alcohol and drugs by 64%, and a greater need for hospitalization for psychiatric illness by 50% [13].

The data from the first 1,000 patients treated in the Georgia Impaired Physician Program of Atlanta are shown in Table 2 [14]. In terms of highest incidence of occurrence in that program, the family and general practitioners headed the list followed by the anesthesiologists. The family practitioner was in solo practice, was rural based, was older and isolated from other physicians, and abused alcohol. The anesthesiologists were younger and used parental narcotics. It is apparent from this table and the maldistribution of occurrence that the two endangered species are now emergency-medicine physicians and cardiothoracic surgeons.

It is of interest that those carrying out emergency work and operations, although only 8% of all US physicians according to the AMA, constituted almost 20% of the first 1,000 patients admitted to this special program. Clearly, this increased incidence is the reflection of being in the combat zone too long with too little real rest.

Is This Burnout?

Little is known about this syndrome in surgeons. The factors associated with burnout in physicians and surgeons appear to be similar. The personality traits present on entering medical school are the same ones that tend to be related to burnout; particularly, the tendency toward depression. Individuals with this syndrome perceive high stress, although in others' eyes, these stresses may not be high at all. Burnout victims almost universally express a marked dissatisfaction with their careers, their marriages, and their general life styles. The burnout personality is inversely related to the perceived control over job characteristics; that is, those who had high control had a very low incidence of burnout. Not related to burnout are sex, age, marital status, medical specialty, practice arrangement, workload, time with patients, or hours worked [15].

In Summing Up, What Price Do We Pay?

We pay with long work weeks that average more than 60 hours. We marry later. We tend to have fewer children. We have a higher than normal rate of divorce. We are at risk for addiction. We do not know yet whether our group has an increased incidence of early death.

Hobbies

Having profiled who we are as achievers in the surgical domain and what prices we must pay, I wish to put forth the notion that these achievements are a continuum motivated by at least two factors: that which is intrinsic in each of you, and that which is extrinsic. By extrinsic, I mean your other self—the personal, the special part outside of surgery; a part that is uniquely yours and a part of the matrix of those with whom you share your life.

A few serious scholars have recognized this need for an avocation outside of the professional life. Sir William Osler, writing in the *British Journal of Medicine*, said the following:

> No man is really happy or safe without a hobby, and it makes precious little difference what the outside interest may be . . . botany, beetles or butterflies, roses, tulips or irises, fishing, mountaineering or antiquities . . . anything will do as long as he straddles a hobby and rides it hard.

The last image of riding a horse as a hobby is frequent. Sayce commented on the rider whose sole mission was his profession. He said, "Transgress the boundaries of scientific evidence and incur the charge of riding a hobby too hard." Newton, like Osler, advocated a hobby.

> Young man, get a hobby; preferably two: one for indoors and one for out. Get a pair of hobby horses that can safely be ridden in opposite directions.

Or this from Coleridge, despairingly:

> Everyone has (to use the cant term of the day) his hobby horse—something that pleases the great boy—for a few hours.

Some have interpreted hobbies in other ways. This quote is from an early thoracic surgeon, Sauerbruch: "Many bottles have I drunk and many women have I loved."

Osler, again: "You will be a better man and not worse practitioner for an avocation." Commenting on home life, Sir Henry Wooten noted:

> Next to no wife and children, your own wife and children are best past time; another's wife and your children—worse; your wife and another's children—worst.

Let me show you a surgeon who had hobbies. John Hunter was the first great English surgeon and scholar who had a large number of interests outside the clinic and his surgery. These included biology, botany, comparative anatomy, geology, mineralogy, numerology, paleontology, and veterinary science.

Heroes

Finally, I want to project the image and importance of heroes in our lives. A hero is not an idol. A hero is a mortal and not necessarily a likable one. However, a hero may be both larger than life in aura and humble, modest, and compassionate. The word *hero* comes from the Greek word *Heros*, a name given to men of superhuman strength, courage, or ability; a person favored by the gods and later regarded as a demigod or immortal. A hero has been described as a man who exhibits extraordinary bravery, firmness, and greatness of soul in connection with a pursuit. He is a man admired and venerated for his achievements and noble qualities. The hero may be a man who forms a subject of an epic, the chief male personage in a poem, play, or story.

What makes a hero? There are three ways. The first is to be a doer of great deeds, and this is out of the classic mold and myth. There are those who accomplish the magnificent. Today, there are few living physicians who qualify as heroes in the public mind. In medical minds, the deeds of Harvey, Lister, Pasteur, Jenner, and Hunter are sterling examples of past heroes.

A second way to be a hero is to have a great character. This stature as a hero depends more on admiration and less on deeds. William Osler and Albert Schweitzer were medical character heroes. Although their deeds were clinically significant and remarkable, it was the sum of their admirable traits, views, and attitudes that made them heroes.

A third way to become a hero is to create one. The artist, actor, and writer may be viewed by the public as having the virtues of the character they created. An example is Marcus Welby. All too often, however, these are images of the ideal; a sum of perfect parts without flaws.

Joseph Campbell, in his book *A Hero of a Thousand Faces*, described a hero as someone who achieved beyond the normal range. But this is not just becoming a Sunday's football star; rather, he described a hero as one who has given his life to some larger purpose—giving his life to others. He described two kinds of heroes: the physical-deed hero who saves a life or sacrifices his for another, and the spiritual hero who has had a psychological adventure that involved a going out and a returning process. Both types necessarily underwent severe trials and had consequent revelations and rewards.

Should we use the term *role models* instead of *heroes*? I think not. This cliché presents the sense of replication and not the maturation of a figure of giant proportions who lives a life that epitomizes high values and great accomplishments. The accomplishments of true heroes are cloaked in a fabric of humanity and humor.

Heroes of an age are mirrors of our times. It is a sad comment that in the last several decades it seems that the American society has lost its love of contemporary heroes. Think for a moment of where our heroes come from today. Do they fit the description of the ancients, the heroes of the great myths? I think not. Our heroes today are short-lived; they are the super athletes and the fleeting stars of television and Hollywood. Are these transient heroes really the mirror of our time? I hope not.

Why Have Heroes?

Heroes are important because once adopted, they symbolize for us an aspiration to improve and to transcend the mundane. The hero or heroine is our example of the value of the struggle to conquer the ordinary. The hero is our beacon; the light that says dream the impossible dream. Heroes imbue in us the notion that hard work, the long hours, the extra mile are all worthwhile. During times of discouragement, they provide an image not to retreat or to become despondent or hesitant.

Let me tell you a few of my heroes. William Stewart Halsted, the innovator of the surgical residency training program in this country at the turn of the century is a hero of mine. For me he is a hero for his original thinking, his lifelong attitude of inquiry, and especially for his remarkable laboratory experiments, which were far ahead of his time.

Alexis Carrell's work over several decades illustrates the dedication to inquiry that yielded remarkable innovations and observations that have served as a basis for modern vascular surgery. There are others, and, after reading the stories of the lives of Cushing, Blalock, and John Gibbon, I hope that you too will see them as true surgical heroes.

In my medical school experience there were several heroes. My physical diagnosis teacher, Dr Elliot Hochstein, could simulate with unique clarity and accuracy the sounds in the chest and in the heart. He had enormous compassion for the ill and a great love and talent for teaching students. His skill in clinical diagnosis transcended the ordinary. He was a hero in the character sense.

There were two basic scientists—one in this country and another in Japan—with whom I worked. The first was a protein chemist, Frank R. N. Gurd, who was a hero for his understanding of the untrained mind—namely, mine. He provided an intellectual breadth and depth that was truly spectacular. He opened my eyes to so many new possibilities and ways of thinking. He was extremely well versed in the humanities and was a member of a local symphony group. He would practice his French horn for his symphonic appearances in the lab with a fresh cup of imported cocoa in front of him. He was a superb teacher and a character hero.

The second was a Japanese pharmacologist. He had performed research at Cornell Medical College for 2 years as part of a senior scientist exchange program and returned to the University of Tokyo as a professor in 1956. He was in charge of a small, specialized, independent laboratory. Here, he studied the role of α and β receptors in the coronary vasculature, the existence of which was being greatly debated in 1958. He is a hero for me for he had a larger-than-life image for his colleagues and trainees and had enormous sense of humor about the fallibility of man. He nearly lost his life near the end of World War II when the Japanese were testing their first jet engine. It was mounted on a platform, atop a tall bamboo tower. Hashimoto was high in the grandstand as a young naval lieutenant assigned to naval intelligence. At ignition the tower collapsed. The engine exploded, and the crowd of the high command in the front rows was awestruck and the audience silent, except Hashimoto, who instantly saw the ludicrousness of the effort, the inevitability of the allied victory, and a basic flaw in the Japanese national character. He began to chuckle until he was laughing nearly uncontrollably and was nearly court-martialled the same day.

He worked with the simplest of instruments but performed elegant, complex experiments. He encouraged and, in fact, required questioning of the data by his Japanese trainees in a society in which it was forbidden for the trainee to question the professor.

The last example of a hero in my life was my teacher of surgery, William Harry Muller, Jr. He was an innovator and a leader in the early days of cardiac surgery. He developed a method for closing atrial septal defects before the advent of cardiopulmonary bypass. He and Frank Damman, a pediatric cardiologist, developed the pulmonary artery banding operation to reduce the torrential excess of pulmonary blood flow in small children. He tried cardiopulmonary bypass clinically using dogs' lungs for oxygenators and used the glass-tube oxygenator in a series of patients with rare survival. The fortitude and courage to continue with these procedures was awesome as was his surgical judgment and technical abilities. His assurance, sense of command, and ability to stay cool in times of great difficulties were all qualities of the hero-leader. I am indebted to him for my training, for his counseling, and for the personal interest he has shown in each of his trainees.

Closing

I have attempted to portray and profile the toughest, most demanding, and most difficult of surgical careers, that of the cardiothoracic surgeon. We are the green berets, the marines, the special forces, the seal teams of the medical profession. We train longer, marry later, have fewer children, and are near retirement before we enjoy these children as young adults. We are still older by the time we see our grandchildren. We have a higher than expected rate of divorce, alcoholism, mental disability, and potentially early death. All of these are hallmarks of a life of prolonged stress.

Despite all of this, a remarkable few have brought more dramatic palliation and cures to disease in a shorter interval on a larger scale than any surgical specialty heretofore.

I have suggested that the psyche of you, the high-performance professionals, and probably those of your loved ones, will be best served if you get a hobby and ride it for all it's worth.

As we have aged singularly and collectively, we have forgotten the importance of the hero in our lives. I hope I have made the case that heroes are needed. Heroes are our beacons in the downpours and in the fog. Heroes enrich and inspire us to persevere and to help us feel that our lives are all worthwhile.

I close with a quotation from a poem published by Robert Frost's daughter in the Rockford Register Republic in 1935:

> Living on the horizon's edge, the growing age of discovery
> for that is where the beauty that lurks in danger and
> defiance and defeat and victory lies.

I congratulate you for who you are and for what you have given of yourselves to others. That is the bond that binds us, *Semper Fi.*

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Assessment of Spinal Cord Blood Supply During Aortic Operations

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There is no question that attempts to identify the critical blood supply of the spinal cord are the key to preventing the most disastrous complication of aortic operations, paraplegia. In this regard, Svensson and associates are to be commended not only for their report in this issue [1], but for their multiple previous investigations on this subject.

Svensson and associates present a unique approach to the monitoring of intercostal spinal cord blood supply using intrathecally placed platinum electrodes. It is of interest to note that as many as one third of the segmental branches of the aorta provide circulation to the spinal cord via reticular arteries. Impressively, the hydrogen-induced current impulses were successful in locating 25 of 28 of these.

See also page 528.

The problem that I have in accepting this technique as potentially clinically applicable is that in only 1 of their 9 experimental animals were they successful in placing the platinum electrodes percutaneously. Moreover, when the electrode is placed blindly, there will be difficulty ensuring that the electrode lies close enough to the anterior spinal artery to ensure that monitoring will be valid.

Another problem is that the technique may be far too sensitive. If the anatomy of the pig should relate in any way to the human, it is conceivable that the surgeon would be faced with the dilemma of having demonstrated that one third of the intercostal or lumbar arteries provide blood supply to the cord. Which of these, if not all, should be reanastomosed to the vascular graft?

It would seem to me that Svensson and associates have gone about the problem backward from the standpoint of developing this technique as a clinically feasible tool. In humans the anterior spinal artery takes its origin from the vertebral arteries, and in most instances, the anterior spinal artery is intact the length of the spinal cord. Under such a circumstance, one could speculate that the patient would not be vulnerable to paraplegia, particularly if decompressive techniques such as spinal fluid drainage or papaverine were used to optimize collateral flow. On the other hand, identifying the critical arteries would be of great value in that group of patients in whom continuity of the spinal artery was not demonstrated. I suggest that the technique might be most practical if it were combined with preoperative catheterization of a vertebral artery with injection of hydrogen-saturated sodium chloride with the monitor placed percutaneously at the lumbar level. If hydrogen could be detected at the low lumbar area, the presumption would be that the anterior spinal artery is intact, and reanastomosis of critical segmental arteries would not be necessary. Conversely, if no hydrogen reached the lower spinal cord, then selected attempts to identify the artery of Adamkiewicz for reanastomosis would be appropriate, whether using arteriography or other techniques such as a hydrogen electrode placed in the thoracic or lumbar area.

I believe Svensson and associates have in their hands an excellent research tool for use in demonstrating spinal cord blood supply, and this technique could conceivably be adopted for clinical use. However, I believe that some other technique that is less invasive will be necessary if monitoring is to be practical and accurate.

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Controversies About Barrett's Esophagus

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The report in this issue by Williamson and colleagues [1] addresses important issues concerning esophagus lined with columnar epithelium. Within the last 15 years, Barrett's esophagus (BE) has become a commonly encountered problem rather than an unusual curiosity as it was regarded in earlier times. Among patients referred for endoscopy to evaluate symptoms of gastroesophageal reflux, more than 15% are found to have 3 cm or more of columnar epithelium lining the peristaltic esophageal tube thus meeting the definition of BE. In parallel, the incidence of adenocarcinoma arising within the esophagus and often associated with Barrett's epithelium is increasing. In our series [2], that of Ellis [3], that of Hameeteman and associates [4], and others, incidence of carcinoma in patients diagnosed with BE is approximately 1 in 75 patient-years of follow-up. Adenocarcinomas arising in the esophagus are now approximately equal in number to squamous cell carcinomas, a remarkable change from reports in previous decades [5].

As noted in the report by Williamson and colleagues and in our own experience, BE occurs significantly more frequently among male patients. The sex and racial distribution of adenocarcinoma arising in Barrett's esophagus is highly unusual with more than 90% of cases occurring in male patients, nearly all of whom are white. The reasons for the apparent increase in the incidence of BE, adenocarcinoma arising in the altered epithelium, and the sex and race distribution are unknown and represent a fruitful field for epidemiologic study.

See also page 537.

A central issue in treating patients with BE is the role of gastroesophageal reflux. The high correlation between abnormal reflux and BE is widely documented, and suggests the theory that severe gastroesophageal reflux damages the squamous mucosa, which is replaced by upward migration of columnar epithelium. However, several observations are difficult to explain by the theory that reflux esophagitis causes BE. The typical metaplastic epithelium of BE is not the normal junctional epithelium of the cardia, although cardial epithelium is certainly seen in some patients with BE. The origin of the metaplastic epithelium with its characteristic goblet cells is unknown. Although the reflux quantitated in patients with BE is frequently high, many patients do not show esophagitis at the abnormally placed squamocolumnar junction. With the frequency with which endoscopy is used to monitor the treatment of esophagitis, one would expect the progression from esophagitis to Barrett's epithelium to be readily and frequently documented. Such cases are rare. At present, reflux and BE must be considered associated conditions without proof of a cause and effect relationship.

Can correction of reflux lead to regression of Barrett's epithelium and reversion to squamous cell mucosa? To my knowledge, a well-documented case of complete regression of Barrett's epithelium after successful medical or surgical treatment does not exist. Measuring regression must be based on an early postoperative endoscopy confirming the continued presence of BE and mapping it with multiple biopsies. The findings at subsequent endoscopies with multiple biopsies are compared with the initial posttreatment endoscopy. Comparison with a preoperative endoscopy is invalid as the mobilization and efforts to lengthen the esophagus during an antireflux repair alter the geometry and location of the squamocolumnar junction. We have seen the squamocolumnar junction relocated several centimeters distally after an antireflux repair. This does not represent regression of the Barrett's epithelium. In a patient with a short segment of BE, it may be difficult to determine whether the esophagus itself is still lined with columnar epithelium postoperatively owing to the surgical distortion. When a Collis gastroplasty is used, the findings are even more difficult to interpret.

A phenomenon seen in approximately one fourth of patients treated successfully by antireflux operation is the spreading of squamous epithelium on top of and into areas previously documented to be lined solely with Barrett's epithelium. We have now found this in patients responding to intensive medical therapy and in 1 patient in whom reflux was diminished by the development of a tight stricture at the site of a Barrett's ulcer. Just proximal to the stricture substantial islands of squamous epithelium appeared. This spread of squamous epithelium is taken to represent healing after reflux is controlled. Whether complete reversion to squamous mucosa can occur over time remains unclear and undocumented. The benefits to the patient from this are not clear, so this finding can not be taken as an indication for an antireflux operation.

Can the progression from metaplastic Barrett's epithelium to dysplasia and on to carcinoma be stopped or reversed by successful treatment of reflux? It is well documented that dysplasia may develop in multiple sites in BE and may vary in degree from site to site. The number of biopsy samples taken bears directly on the probability that dysplasia will be diagnosed. Accordingly, it is nearly impossible to state with certainty that dysplasia is not present preoperatively in a patient in whom adenocarcinoma subsequently develops. To approach the

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question as to whether dysplasia develops after an antireflux operation or whether preexisting dysplasia is already a neoplastic process that progresses, some minimum standard number of biopsies must be agreed on by investigators. Our current practice is to take two biopsy specimens at differing locations from each centimeter of esophagus involved with BE.

Another problem is the interpretation of biopsy material. The grading of Barrett's epithelium as indefinite for dysplasia, low-grade dysplasia, or high-grade dysplasia represents an interpretive judgment by a pathologist. For research studies, slides should be reviewed independently by several pathologists experienced with the interpretation of Barrett's epithelium. The epithelium adjacent to ulcerations is particularly difficult to evaluate because of regenerative and inflammatory responses.

Like Williamson and colleagues, we have patients in whom mild dysplasia was diagnosed preoperatively and not found on subsequent postoperative endoscopies. We, like Williamson and colleagues, have patients in whom adenocarcinoma developed after a documented successful antireflux repair but who had at least mild dysplasia in some preoperative biopsy specimen. It remains unclear whether antireflux operation can prevent the development of dysplasia in Barrett's epithelium. A more thorough study of larger numbers of patients is necessary to address this question. It does seem clear that uncertainty as to the presence of dysplasia preoperatively and the potential for dysplasia to progress to frank carcinoma require patients undergoing surgical or medical treatment for BE to remain under long-term surveillance.

In all reports addressing these questions, the matter of documenting control of reflux is critical. Williamson and colleagues acknowledge that not all patients in their series were completely studied by manometry and pH reflux testing preoperatively and postoperatively, and the more sensitive prolonged pH monitoring studies were not used. It is well known that patients with BE are less susceptible to the symptom of heartburn so that the symptomatic evaluation of the success of treatment is not particularly valid in these patients. Objective evidence of the degree of success in controlling reflux must be obtained when the relationship of preventing reflux to regression of BE or prevention of carcinoma is assessed.

Our present practice for managing patients with BE includes multiple preoperative biopsies, prolonged pH monitoring, and manometry. Indications for antireflux operation are similar to those recommended by Williamson and colleagues, that is, the persistence of ulcerative esophagitis that does not heal on medical therapy, reflux symptoms not responsive to medical therapy, and reflux complications including nonhealing Barrett's ulcers, stricture, and aspiration. Once antireflux operation is done, the patient has an early postoperative endoscopy to replot the extent of the Barrett's epithelium and an early postoperative manometry and pH test to document the initial success of the operation. One, 2, and 5 years after the operation, repeat endoscopy with multiple biopsies and pH and manometry studies are recommended. Annually, an esophageal brush cytology study is performed as a surveillance for the detection of malignant cells.

For the present, we agree with the conclusions of Williamson and colleagues that the indications for antireflux operation in patients with BE should be the same as for other patients with reflux esophagitis and that such patients require ongoing surveillance to detect neoplastic changes at a time when they may still be curable.

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Myocardial Energetics and the Postischemic Heart

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Myocardial "stunning" has been defined as the persistence of myocardial dysfunction in previously ischemic myocardium, despite restoration of blood flow. This phenomenon, also referred to as postischemic myocardial dysfunction, can affect the clinical course of patients spanning the spectrum of coronary artery disease [1]. For example, some patients with episodes of either exertional or unstable angina may suffer from prolonged bouts of myocardial dysfunction despite termination of ischemia by antianginal therapy and the absence of myocardial infarction. Some patients may suffer circulatory compromise after a myocardial infarction despite relatively early revascularization by thrombolytic therapy or bypass grafting. Withdrawal of cardiopulmonary bypass may be difficult initially in some patients after a prolonged heart operation, yet ventricular function may improve without any specific therapy over the course of days to weeks. In each case, postischemic myocardial dysfunction could be the explanation for ventricular pump failure despite the absence of ongoing ischemia or myocardial necrosis.

It has come to be appreciated that the postischemic myocardium is energetically inefficient. This notion is based on observations from several laboratories that myocardial oxygen consumption (MVO_2) is disproportionately high for the appreciably depressed level of mechanical work performed in stunned as compared with normal myocardium [2, 3]. Although much is known about postischemic myocardial dysfunction, relatively little is known about the time course, extent, and consequences of postischemic metabolic inefficiency of the myocardium. It can be hypothesized, however, that postischemic myocardial dysfunction results from abnormal coupling between force generation and chemical energy utilization in muscle cells. It is therefore important to elucidate the mechanisms of this inefficiency as a first step toward devising means of prevention and treatment.

See also page 543.

In previous studies of postischemic myocardial energetics, myocardial efficiency has been defined as a ratio between mechanical work and MVO_2, measured at only one hemodynamic loading condition in a given heart. However, the information that can be gained about abnormalities in contractile performance or various energy handling processes by this approach is limited. Important information might be ascertained about the energetics of stunned myocardium by examining the interrelations between contractile state, mechanical work, and oxygen consumption measured over a range of hemodynamic loading conditions within each heart. In 1979, Suga and colleagues [4] introduced a new framework for examining myocardial energetics at the ventricular level that takes advantage of this approach.

This framework is based on the relationship between MVO_2 and the pressure–volume area (P-VA). Pressure–volume area is the area on the pressure–volume diagram circumscribed by the end-diastolic pressure–volume relation, the end-systolic pressure–volume relation (ESPVR), and the systolic portion of the pressure–volume trajectory during the beat (Fig 1, top panel). Pressure–volume area represents the sum of the external stroke work (area within the pressure–volume loop) plus a quantity of potential energy "stored" in myocardial muscle at end systole [4]. It has been hypothesized, therefore, that P-VA represents the total mechanical energy generated by the left ventricle on a contraction [4]. Support for this hypothesis is provided by the observations that (1) P-VA is linearly correlated to MVO_2 (Fig 1, bottom panel) and (2) the MVO_2–P-VA relation is fairly insensitive to changes in ventricular loading conditions [5].

To the extent that P-VA indexes total mechanical energy, the slope and MVO_2-axis intercept of the MVO_2–P-VA relation index various aspects of myocardial energetics. The intercept value is the MVO_2 measured from the mechanically unloaded heart. In ideal terms, this "unloaded MVO_2" can be thought of as the sum of basal metabolic MVO_2 plus the energy required for excitation-contraction coupling, primarily required for calcium cycling by the sarcoplasmic reticulum [6]. Basal metabolic MVO_2 has been shown to be independent of contractile state in hearts arrested by hyperkalemic solutions. The MVO_2 for excitation-contraction coupling can be changed by interventions that alter the amount of calcium cycling (eg, inotropic agents) or, in principle, any action that would change the ratio between adenosine triphosphate production and MVO_2 (P:O ratio). The slope of the MVO_2–P-VA relation, mathematically defined as the change in MVO_2 for a given change in P-VA, has been proposed to reflect the "efficiency" of pressure generation by the ventricle [4, 6]; the lower the slope, the greater the efficiency. The value of the slope could be influenced by any intervention that would change the relationship between force generation and adenosine triphosphate utilization by the myofilaments or change the P:O ratio. Thus far, there has been no intervention demonstrated to alter the slope of the MVO_2–P-VA relationship, though there are a number of as yet untested conditions under which the slope may be changed.

The "inefficiency" of the postischemic myocardium could result from ischemic or reperfusion-mediated injury

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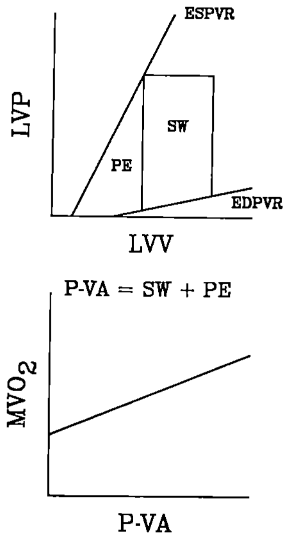

Fig 1. (Top) Pressure–volume area (P-VA) *represented as the sum of the external stroke work* (SW) *plus a quantity of potential energy* (PE) *stored in the myocardial muscle at end systole. (Bottom) Linear correlation between P-VA and myocardial oxygen consumption* (MVO_2). (EDPVR = *end diastolic pressure–volume relation;* ESPVR = *end-systolic pressure volume relation;* LVP = *left ventricular pressure;* LVV = *left ventricular volume.)*

to one or several energy-using or force-generating processes within myocytes. Some possibilities include (1) uncoupling of mitochondrial oxidative phosphorylation, (2) increased basal metabolism, (3) alterations in energy required for calcium metabolism, and (4) increased adenosine triphosphate requirements for force generation by the myofilaments. Previous studies have addressed several of these possibilities. First, P:O ratio is normal in the stunned myocardium indicating that mitochondrial function is intact (ie, no evidence of mitochondrial uncoupling). Second, it has been shown that basal metabolic MVO_2 is not significantly different in normal and stunned myocardium [3]. Third, there is evidence of decreased myofilament sensitivity to Ca^{2+} and increased peak intracellular calcium during a beat in stunned as compared with normal myocardium [7, 8]. Finally, it has been shown that calcium-transport mechanisms of the sarcoplasmic reticulum are impaired in stunned myocardium [9]. It is noteworthy for the present discussion that the energetic consequences of these latter observations regarding calcium metabolism in terms of the relation between mechanical work performed and oxygen consumption have not been evaluated.

In view of their physiological interpretations, examination of alterations in the variables of the MVO_2–P-VA relationship may offer new and valuable insights into mechanisms responsible for the energetic inefficiency of stunned myocardium. Furthermore, simultaneous with the measurement of the MVO_2–P-VA relationship, information is also obtained regarding the ESPVR, which offers detailed information regarding the contractile state of the heart. With these concepts in mind, Furukawa and colleagues [10] have measured the MVO_2–P-VA and ESPVRs under control and stunned conditions in ovine hearts.

Briefly, Furukawa and colleagues instrumented open chest sheep to measure left ventricular volume by sonomicrometry, left ventricular pressure, oxygen saturation of blood in the coronary sinus, and coronary flow. Alterations in hemodynamic loading conditions were achieved by balloons placed in the aorta. Measurements were made for determination of the ESPVR and the MVO_2–P-VA relation at baseline conditions. The sheep were then cannulated and placed on cardiopulmonary bypass at 37°C for 25 minutes with the aorta cross-clamped. After reflow, the hearts were defibrillated and supported by continuous infusion of lidocaine and epinephrine until ten minutes before repeat measurements of MVO_2–P-VA and ESPVRs; the average duration of reflow before making measurements was 84 minutes. The results showed that the slope of the ESPVR decreased in most hearts after the ischemic period while the volume axis intercept increased. Both of these findings indicate a reduced contractile state. The slope of the MVO_2–P-VA relation was increased in the stunned conditions as compared with the initial control conditions, whereas there was no change in the MVO_2-axis intercept.

If the P:O ratio and basal metabolic MVO_2 are normal in the stunned myocardium, as suggested by the results noted previously, then the findings of Furukawa and co-workers would suggest that one factor contributing to the metabolic inefficiency of the stunned myocardium relates to alterations in the transduction of chemical energy into mechanical energy, presumably by the myofilaments. The finding of no change in MVO_2 intercept is unexpected in view of the previous data showing increased levels of calcium cycling in stunned myocardium. On the other hand, the findings may complement the idea of decreased myofilament affinity for calcium and further localize the myofilaments as a major site of postischemic myocardial dysfunction.

In summary, the mechanisms that underlie the inefficiency of the postischemic myocardium are not understood. Although it has been recognized that myocardial function can be depressed for days or weeks after an ischemic episode, the duration, degree of recovery, and consequences of myocardial inefficiency are not well understood. In principle, the MVO_2–P-VA relationship may be useful in providing clues as to which aspects of energy handling processes are targeted by ischemic or reperfusion-mediated injury. The results of such studies could not speak to the mechanisms by which the injury itself occurs (eg, free radicals), but may suggest ways of dealing with ischemic and reperfusion injury in several common clinical settings. In this regard, the results of the study by Furukawa and colleagues showing an increased slope of the MVO_2–P-VA relationship in postischemic myocardium provide an intriguing clue as to why the stunned myocardium is inefficient and deserve further investigation.

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Preliminary Report of Localization of Spinal Cord Blood Supply by Hydrogen During Aortic Operations

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One source of paraplegia after aortic operations is the failure to reattach the spinal cord blood supply, the origins of which are not evident at operation. This report is concerned with a rapid new method of identifying these vessels intraoperatively. In 9 pigs, a specially designed catheter with platinum and stainless steel electrodes was inserted intrathecally. Saline solution saturated with hydrogen was injected sequentially into arterial ostia at T-15 to L-4 inclusive, and the generated current impulses from the conditioned platinum electrode were recorded. Of 90 potential segmental arteries supplying the spinal cord, 28 gave rise to spinal radicular arteries. Hydrogen-induced current impulses correctly located 25 of the radicular arteries and all those larger than 180 μm in diameter. When injected with indigo carmine, the vessels localized by the hydrogen-induced current impulses filled the entire anterior spinal artery from the low thoracic to the sacral region, whereas injection of the other vessels did not show filling. After refinement and testing for safety, this method has been employed clinically to rapidly localize and reattach routes of critical cord circulation.

(Ann Thorac Surg 1990;49:528–36)

The incidence of paraplegia in patients after replacement of the descending thoracic or thoracoabdominal aorta is reported to be 40% or greater [1–3] despite available modern techniques. Most of these techniques, which include the application of distal aortic perfusion, deep hypothermia, oxygen radical scavengers, allopurinol, calcium-channel blockers, and cerebrospinal fluid drainage alone without other adjuncts have, in our experience, been ineffective in adequately preventing paraplegia [1–2, 4–10]. The likely reason is that the etiology of paraplegia is multifactorial, and thus more than one strategy is needed for the effective elimination of paraplegia.

For editorial comment see page 522.

The major causes of paraplegia that should be addressed are the degree and the duration of ischemia during aortic cross-clamping [1, 8, 10], and ischemia occurring after unclamping because of failure to reattach to the aortic graft those segmental intercostal and lumbar arteries that supply the spinal cord radicular arteries, including the arteria radicularis magna (ARM) (artery of Adamkiewicz) [10–16]. The latter point poses a dilemma in that, in the interest of reducing the duration of ischemia during aortic cross-clamping, the general practice is to reattach none or only a few of the segmental arteries [1, 2, 10]. Clearly, if there is a failure to reattach the segmental arteries that supply the spinal cord (not all of them do [Fig 1]), this will result in the spinal cord being deprived of its normal source of blood flow when the aorta is unclamped.

Amelioration of the depth and duration of ischemia during aortic cross-clamping has been addressed by the use of intrathecal administration of papaverine hydrochloride combined with cerebrospinal fluid drainage [1, 3, 7, 10, 14, 15], which successfully prevented immediate postoperative paraplegia, despite long cross-clamp times, in a series of 34 patients followed up prospectively by one of us (L.G.S.) [15]. Nonetheless, a few days after operation, delayed paraplegia developed in 1 patient and delayed paraparesis developed in 2 patients when the effect of the papaverine would have worn off and the spinal cord was once again dependent on its normal source of blood supply from segmental arteries [1, 15]. In none of these patients were the segmental arteries reattached. Both patients with paraparesis recovered.

The diverse and complex anatomy of the spinal cord blood supply in nonhuman primates and humans [16] has resulted in a major problem in determining which of the 13 or more pairs of segmental vessels (intercostal or lumbar arteries) arising from the aorta supply the spinal radicular arteries (Fig 2; see Fig 1). Although preoperative highly selective angiography has been advocated [3], the risks involved [16], particularly of paraplegia complicating the procedure both in humans and nonhuman primates [13, 16, 17], has made this an unpopular procedure except in expert hands [3].

In this preliminary report, we present a new hydrogen-induced current impulse (HICI) technique to identify the spinal cord blood supply. This is a qualitative method

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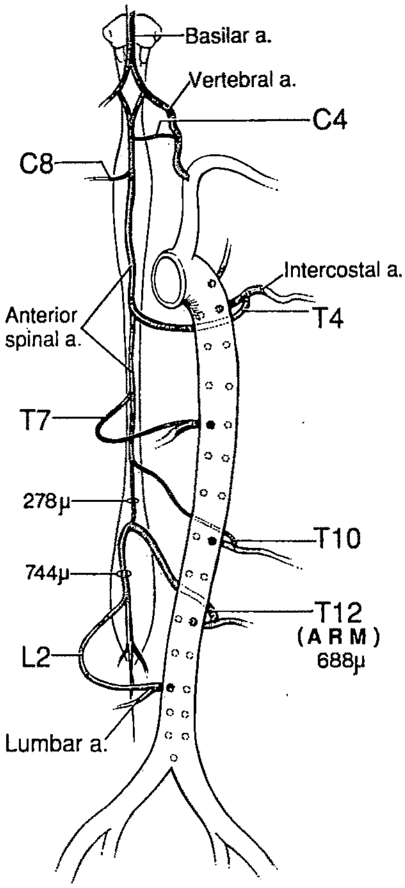

Fig 1. Statistical composite of spinal cord blood supply in primates. The sites of the radicular arteries reflect the most common levels of origin and arrangements found on anatomical dissections. The sizes of the arteries are those of nonhuman primates. The equivalent sizes in humans are: arteria radicularis magna (ARM)*, 871 μm; anterior spinal artery above the* ARM*, 231 μm; and anterior spinal artery below the* ARM*, 941 μm. (After Svensson et al [16].)*

with the potential for allowing the surgeon to determine intraoperatively which segmental vessels can be safely oversewn during aortic operation and which vessels should be reanastomosed to the aortic graft to avoid depriving the spinal cord of its vital blood supply. We also present a new method of eliciting spinal motor evoked potentials directly from the spinal cord.

Hydrogen in solution, when in contact with platinum, produces a weak current that can be measured by a sensitive ammeter [18, 19]. For our new technique, we hypothesized the following: First, because hydrogen readily crosses tissue membranes [18], if injected into a segmental artery supplying the anterior spinal artery, it would readily traverse the anterior spinal artery wall and the spinal cord pia mater and thus be detected by a platinum electrode lying on the surface of the spinal cord, as opposed to being detected by an electrode within the neural tissue of the spinal cord. Second, that if the platinum electrode was close enough to the anterior spinal artery, the response to a hydrogen injection would be almost immediate. Third, that if an HICI was elicited, this would indicate that a segmental artery supplying the spinal cord had been injected. The results of this new technique in a series of porcine experiments and the application of these findings with respect to humans are discussed.

Material and Methods

Nine domestic pigs, 27.5 to 32 kg in weight (mean weight, 29.7 ± 1.5 kg), were sedated with ketamine hydrochloride (20 mg/kg), intubated, and ventilated by a Harvard ventilator with halothane, oxygen, and nitrous oxide. Catheters were placed through the femoral vein into the right atrium for central venous pressure monitoring and fluid administration, through the femoral artery into the infrarenal abdominal aorta for distal aortic pressure monitoring, and through the brachial artery into the proximal aorta for proximal aortic pressure monitoring. The electrocardiographic tracing was monitored continuously on a Beckman oscilloscope and recorder.

The animals were turned on their right sides, and a specially constructed spinal catheter with platinum and stainless steel electrodes was introduced intrathecally either by a lumbar puncture (n = 1) or a laminectomy (n = 8) into the spinal canal alongside the spinal cord. The electrodes were connected respectively to a sensitive ammeter and an electrical stimulator for elicitation of motor evoked responses and potentials. A potentiating current was applied to the platinum electrode to condition it as described by others [18–20].

A thoracoabdominal incision was performed, and the aorta was cross-clamped above the diaphragm and at the aortic trifurcation. In 4 animals, an intrathecal injection

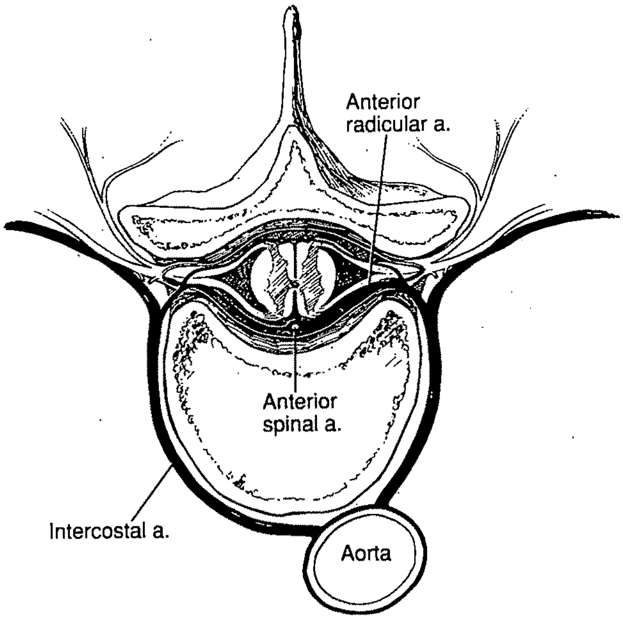

Fig 2. Arrangements of segmental intercostal or lumbar arteries and the origin of radicular arteries coursing to the anterior spinal artery. Note that the radicular arteries cannot be seen intraoperatively unless the overlying tissues are dissected off the vertebral bodies, including the psoas muscle in the abdomen. Both radicular arteries, one radicular artery, or no radicular artery can be present at any specific level.

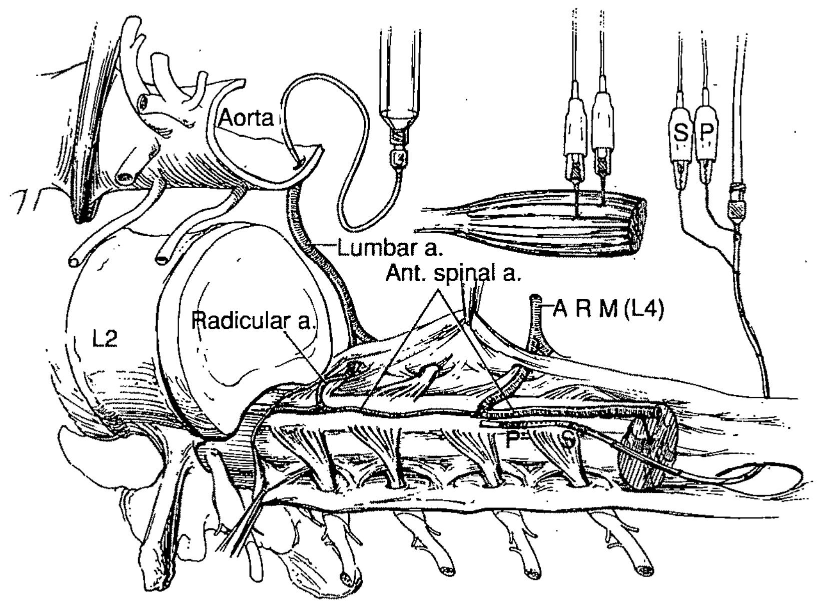

Fig 3. Experimental procedure. Note that the proximal aorta, lying on the vertebral bodies (L1-L2), has been cross-clamped. The more distal aorta has been opened, and saline solution saturated with hydrogen has been injected into the lumbar artery giving off a radicular artery that supplies the anterior spinal artery (Ant. spinal a.) *and spinal cord. The more distal platinum electrode* (P) *detects the hydrogen-induced current impulse.* (ARM = *arteria radicularis magna;* S = *stainless steel electrode.)*

of papaverine was administered before aortic cross-clamping. The aorta was opened and segmental arteries, that is, the intercostal and lumbar arteries, were sequentially injected by hand with 10 mL of saline solution saturated with hydrogen (Fig 3). Each injection procedure took approximately 30 seconds to one minute to perform, and the HICI response was noted within 5 seconds of injection.

The HICI produced was monitored and recorded to see if any hydrogen reached the spinal cord. Figure 4 shows an example of an HICI tracing. If an HICI was elicited, the artery was noted for later dye studies. After all the arteries from T-15 to L-4 inclusive had been injected, those arteries that had been shown to be associated with an HICI were reinjected to confirm the current response. The animals were then given a lethal concentrated solution of potassium chloride.

Indigo carmine was injected into the ostia of segmental arteries as follows: In 3 animals, the dye was injected into the HICI-localized arteries and then the segmental arteries were dissected out from the aorta to the spinal radicular arteries on the spinal cord. In 2 animals, the segmental arteries were dissected free, the anterior vertebral column was removed, the spinal cord was exposed, and the segmental arteries were then injected. In 1 animal, the arteries not localized by the HICI technique were injected. In another 3 animals, the nonlocalized arteries were injected, the cord was exposed, and then the localized arteries were injected. In none of the animals were the segmental arteries damaged or dissected by the injections on gross examination.

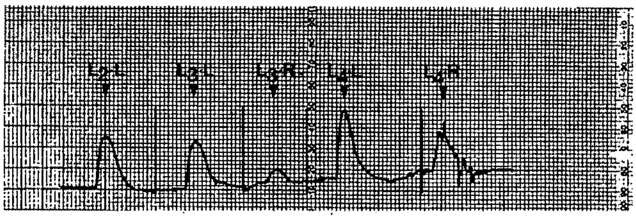

Fig 4. Tracing of hydrogen-induced current impulses in animal 3. (L = *lumbar level;* ·L *and* ·R = *left and right lumbar artery ostium, respectively.)*

The accuracy of the HICI technique in detecting the spinal radicular arteries compared with that of the anatomical dissections of the dye-stained arteries was then correlated. The diameter of the anterior spinal artery above and below the ARM and of the other radicular arteries was measured with a vernier using magnification. Spinal motor evoked potentials were elicited in 5 pigs using a Metraco stimulator and evoked potential system, and the motor evoked potentials were viewed on a Kikusui CO55020 oscilloscope and recorded continuously on an Apple IIE computer for later analysis of the waveform. Electrode deviations in the right and left hip extensor muscles were recorded.

The electromyographic activity was filtered using a low-frequency filter of 0.5 Hz and a high-frequency filter of 3,000 Hz. Full-scale gain was initially set at ±800 mV. Stimulus intensity was gradually increased to 10% above the threshold level using 100-ms pulse duration. Usually single responses were measured. At times, up to ten spinal motor evoked potentials were averaged, at a stimulus rate of 9.7 per second. Spinal motor evoked potentials were elicited at a maximum of five-minute intervals. When the evoked potentials markedly decreased in amplitude or disappeared, larger stimulus voltages were used to try to elicit a response.

The animals received humane care in compliance with the "Guide for the Care and Use of Laboratory Animals" published by the National Institutes of Health (NIH Publication No. 85-23, revised 1985).

Results

Hydrogen-induced current impulses were detected in every animal and occurred immediately after injection of

Table 1. Hydrogen-Induced Current Impulse–Localized Spinal Radicular Arteries and Those Identified During Anatomical Dissections

Animal No.	Ostia Localized[a]	Radicular Arteries Identified and Diameter (μm)[b,c]	Anterior Spinal Artery Diameter (μm)		Arteria Radicularis Magna and Diameter (μm)
			Above[d,e]	Below[d,f]	
1	Rt L-2	Rt L-2, 250	63	250	Lt L-5, 250
2	Lt L-2, Lt L-4	Lt L-2, 200; Lt L-4, 225	155	456	Lt L-5, 300
3	Lt L-2, Lt L-3, Rt L-3[a] Lt L-4, Rt L-4	Lt L-2, 325; Lt L-3, 250 Lt L-4, 430; Rt L-4, 300	275	605	Lt L-4, 430
4[g]	L-2, Lt L-4	Rt L-1, 180[b]; Rt L-2, 351; Lt L-4, 450	450	810	Lt L-4, 450
5[g]	L-2, L-4,	Rt L-2, 275; Rt L-4, 325	250	550	Lt L-5, 355
6	L-2, Lt L-4, Rt L-4	Lt L-1, 180[b]; Lt L-2, 205; Lt L-4, 275; Rt L-4, 300	275	456	Rt L-5, 275
7[g]	Lt L-2, Lt L-3, Lt L-4	Lt L-1, 180[b]; Lt L-2, 351; Lt L-3, 405; Lt L-4, 455	430	635	Rt L-5, 385
8[g]	Lt T-15, Lt L-1, Lt L-2, Rt L-3, Lt L-4	Lt T-15, 380; Lt L-1, 380; Lt L-2, 200; Rt L-3, 430; Lt L-4, 550	735	760	Rt L-5, 550
9	Lt L-3, Lt L-4, Rt L-4	Lt L-3, 180; Lt L-4, 225; Rt L-4, 250	225	450	Rt L-5, 500

[a] There were 26 ostia localized with one false-positive result. [b] There were 28 arteries identified with three false-negative results. [c] The radicular artery diameters were significantly greater in the papaverine-treated animals ($p < 0.025$; Mann-Whitney U test = 107) compared with the nontreated animals. [d] Above and below arteria radicularis magna. [e] Significance: $p = 0.05$ between papaverine-treated animals and nontreated animals. [f] Significance: $p = 0.02$ between papaverine-treated animals and nontreated animals. [g] This animal received papaverine.

L = lumbar level; Lt = left; Rt = right; T = thoracic level.

the hydrogen solution. Furthermore, in every animal, radicular arteries supplying the spinal cord were correctly localized on postmortem examination (Table 1). Moreover, these localized radicular arteries were shown to perfuse the entire spinal cord from the lower thoracic segment to the caudal tip of the spinal cord with dye injections. None of the nonlocalized arteries injected with dye filled the anterior spinal artery. Of interest, in every animal, the anterior spinal artery was also found to be continuous over these segments.

Of 90 potential segmental arteries supplying the spinal cord radicular arteries, some arising as pairs from a common aortic ostium, 28 supplied the spinal cord on postmortem examination, and of these, 25 (89%) were correctly localized as supplying the spinal cord by hydrogen injections. The three radicular arteries that were not identified were 180 μm in size and at least three vertebral levels further away from the platinum electrode (see Table 1). However, by increasing the sensitivity of the technique, we would probably have been able to detect these vessels.

In 1 animal (animal 3; see Table 1), a small HICI measuring 16% of the highest recorded amplitude in this animal was noted, but no radicular artery was identified on the right at that level, although large radicular arteries, which had been correctly localized, were present on the left and at the segments immediately adjacent to it. Collateral blood flow to the correctly localized adjacent radicular artery may have accounted for this weak response. In this same animal, in which five radicular arteries were localized, there was a direct correlation between the size of the radicular artery and the height of the recorded current peak impulse ($r^2 = 0.98$; standard error of the mean, 0.147; $p < 0.01$) (Fig 5). The initial upstroke height of the HICI peak appears to be the best indicator of vessel size rather than the downstroke or area under the curve; however, this requires further research.

Figures 6 and 7 show spinal cord specimens and the HICI-identified arteries from animals 2 and 4, respectively. Figure 8A shows a spinal cord specimen (animal 8) after the lumbar arteries not supplying the spinal cord, as determined by the hydrogen-saline technique, were injected with indigo carmine. None of the dye reached the anterior spinal artery. Figure 8B shows the same spinal cord after the localized lumbar arteries were dissected free to their respective vertebral foramens where the spinal radicular arteries arise. The lumbar artery ostia were then injected with dye. Note that the radicular arteries and the continuous anterior spinal artery were then filled with dye for the entire extent from the lower thoracic spinal

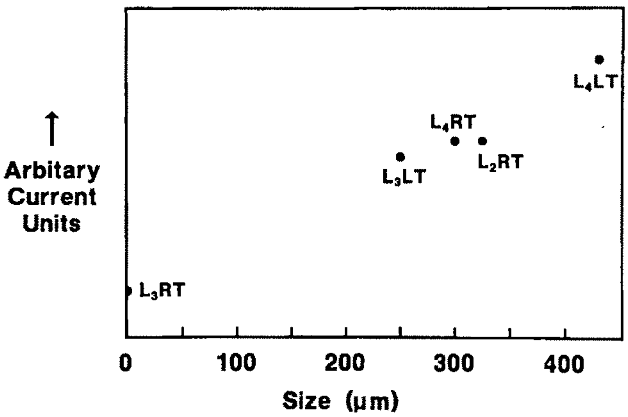

Fig 5. Linear correlation between radicular artery size and hydrogen-induced current impulse amplitudes in animal 3 ($r^2 = 0.098$; p < 0.01). See Table 1. (L = *lumbar level;* LT = *left;* RT = *right.)*

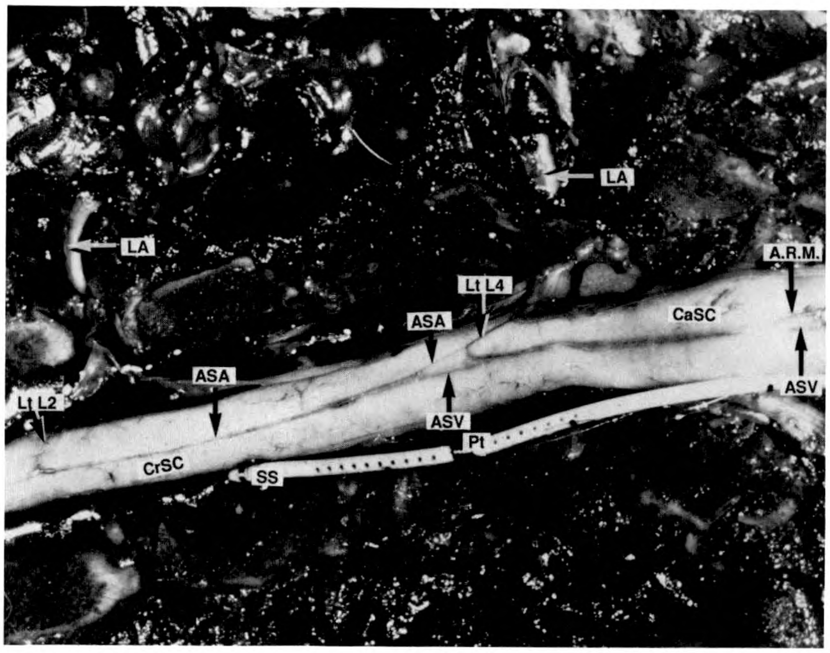

Fig 6. Spinal cord specimen from and identified radicular arteries in animal 2. The catheter with the electrodes is 1.6 mm (1,600 μm) in diameter. Arterial diameters are given in Table 1. (×2 before 38% reduction.) (ARM = *arteria radicularis magna;* ASA = *anterior spinal artery;* ASV = *anterior spinal vein;* CaSC = *caudal spinal cord;* CrSC = *cranial spinal cord;* L = *lumbar artery level;* LA = *lumbar artery;* Lt = *left;* Pt = *platinum electrode;* SS = *stainless steel electrode.)*

cord down to the sacrum. In every other animal in which the localized lumbar artery ostia were injected with dye, both the radicular arteries and the anterior spinal arteries were filled with dye.

The spinal motor evoked potential amplitudes progressively decreased with time after cross-clamping, and by 18.5 minutes (standard deviation, ± 3.1 minutes), they were unrecordable or reduced to less than 10% of baseline values. In 3 animals in which responses were evoked at one-minute intervals, this decrease was observed to be briefly preceded by an initial increase in amplitude of the responses. As expected, the systolic blood pressure increased after aortic cross-clamping (before, 92 ± 10.2 mm Hg [mean ± standard error of the mean], and after, 108 ± 12.3 mm Hg; $p < 0.05$) as did the central venous pressure (before, 16 ± 1.8 mm Hg, and after, 19 ± 3.3 mm Hg; $p < 0.05$) [8, 15].

Comment

This study has shown that the critical segmental lumbar or intercostal arteries that supply the spinal cord can be

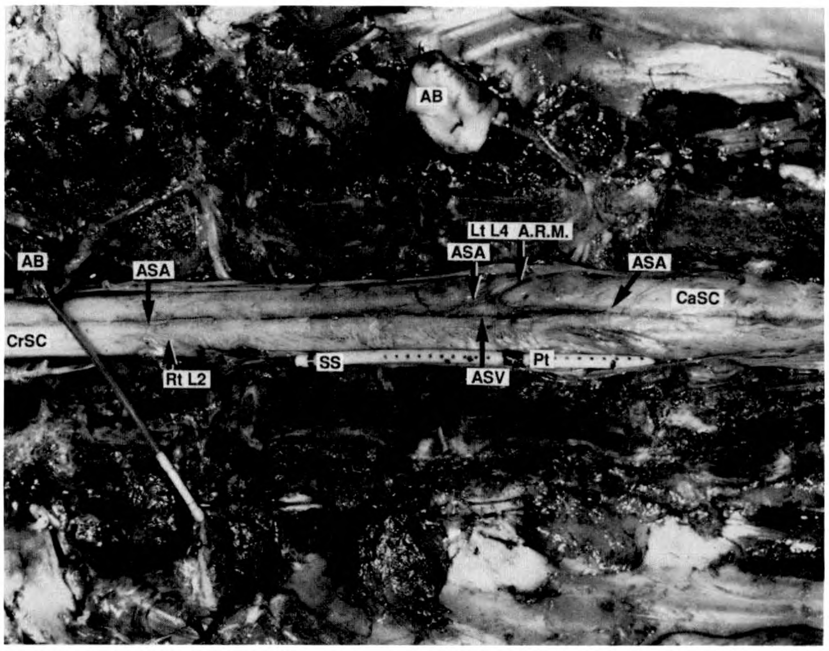

Fig 7. Spinal cord specimen from and identified radicular arteries in animal 4. Arterial diameters are given in Table 1. (×1.5 before 38% reduction.) (AB = *aortic button;* Rt = *right; other abbreviations are the same as in Figure 6.)*

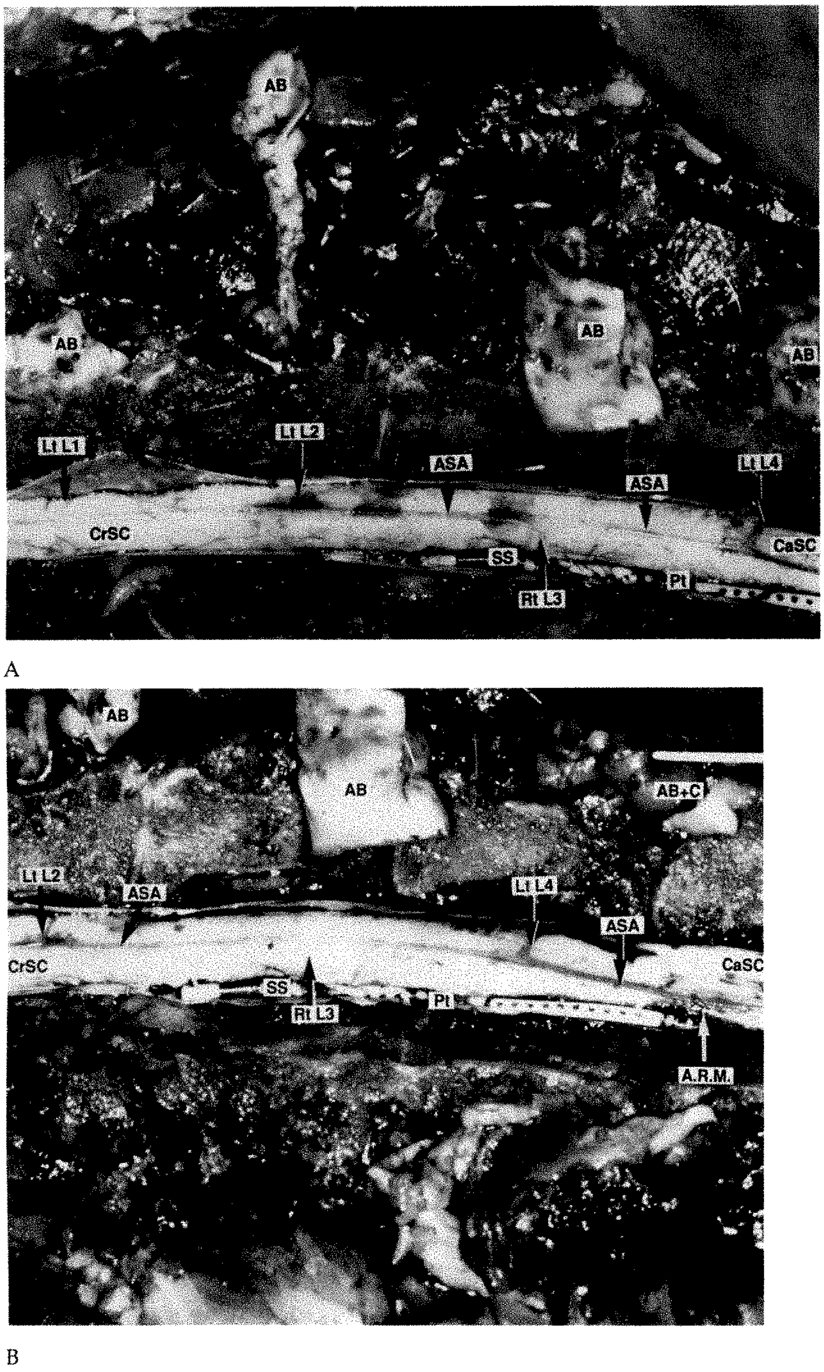

Fig 8. (A) Spinal cord specimen from animal 8 shows the failure of dye to reach the spinal cord when injected into the segmental arteries not identified as supplying the spinal cord. (×1.7 before 38% reduction.) (B) After injection of dye into the segmental arteries shown by hydrogen-induced current impulses to supply the spinal cord, the spinal radicular arteries and the entire continuous anterior spinal artery are opacified. (×1.8 before 38% reduction.) (C = catheter for dye injection; all other abbreviations are the same as in Figure 7.)

accurately and rapidly localized intraoperatively by the use of HICIs. Thus, we propose that if these localized segmental artery ostia can be successfully reanastomosed to a new aortic graft, then the blood supply should be reestablished to the spinal cord. With this new technique, with further testing in live animal experiments for safety and influence on postoperative paraplegia rate, and with appropriate refinement for clinical use, it is likely that the incidence of postoperative paraplegia, particularly delayed paraplegia, can be reduced in humans.

Our three hypotheses concerning our technique have been confirmed by the findings. First, hydrogen readily crosses the intervening membranes between the blood and the platinum electrode placed on the spinal cord, as opposed to being placed in the neural tissue. Second, the HICI is immediately detected in the recording platinum electrode. Third, the ostium of a segmental artery that supplies a spinal radicular artery and is injected with hydrogen-saturated saline solution can be accurately localized to supply the anterior spinal artery.

Previously, the hydrogen polarographic technique was used to measure tissue blood flow [18, 19] by insertion of a platinum probe into the tissue under study. This was done by achieving a constant concentration of hydrogen

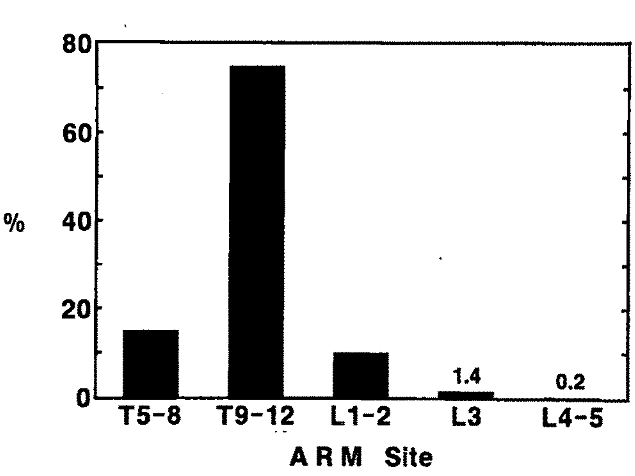

Fig 9. Approximate frequency of origin of the arteria radicularis magna (ARM) according to vertebral levels in humans. (L = lumbar artery level; T = thoracic artery level.) (After Svensson et al [16].)

and a steady-state in the tissues [18, 19]. The hydrogen source was then discontinued, and the rate of tissue clearance of hydrogen was used to calculate the tissue blood flow by the Fick principle [18, 19].

In these experiments, however, we could not measure spinal cord blood flow per se in milliliters per 100 grams of tissue per minute with our present methods probably because the hydrogen was injected under pressure and the sensing electrode was on, rather than in, the spinal cord tissue. Furthermore, the rate of decline of HICI appeared to be dependent on the distance the electrode was from the anterior spinal artery. Thus, many of the elicited HICI peaks were asymmetrical. Nevertheless, the source of the spinal cord blood supply was accurately localized.

Although we [1, 16] have previously noted on lateral roentgenograms that intrathecal catheters lie anterior to the spinal cord in humans, in these experiments, if the platinum electrode was not situated exactly alongside the anterior spinal artery, an HICI response was still measurable, even if the electrode was alongside the ligamenta denticulata of the spinal cord. Furthermore, in our experience, radicular arteries were localized up to five spinal cord vertebral segments from the platinum electrode. Nonetheless, we did try to position the electrode as distally as possible on the anterior spinal cord, as we [7, 16] have previously shown that, during aortic cross-clamping, blood flow is probably in a cranial to caudal direction in the anterior spinal artery. Using two platinum electrodes, this appeared to be confirmed in 2 animals. Thus, any hydrogen injected upstream from the catheter is detected further caudally.

We therefore speculate that as we have placed the tip of intrathecal spinal catheters as high as vertebral level T-10 in humans, approximately the level of spinal cord segments L-2 to L-3, then those radicular arteries, including the largest, the ARM, that usually arise in 90% of patients between T-8 and L-3 (Fig 9) [16] should be able to be accurately localized. Although three very small radicular arteries at the furthest cranial distance from the platinum electrode were missed in these animal experiments, in similar-weight nonhuman primates (*Papio ursinus*), we [16] have shown that the ARM (mean, 688 μm) and anterior spinal artery (means: above ARM, 278 μm; and below ARM, 744 μm [see Fig 1]) are considerably larger than those in untreated pigs (means: ARM, 391 μm; above ARM, 199 μm; and below ARM, 443 μm [see Table 1]) and therefore are less likely to be missed. Furthermore, in our human adult spinal cord dissections, both the ARM (mean, 871 μm) and the anterior spinal artery (means: above ARM, 231 μm, and below ARM, 941 μm) were generally even larger [16].

Although the purpose of this study was not to statistically analyze the comparative anatomy of human, nonhuman primate, canine, and porcine spinal cords, we did examine injected postmortem specimens of both canine and porcine spinal cords with a view to their suitability for this study. We selected the pig for these experiments because the ARM (artery of Adamkiewicz) arises fairly consistently from the lower lumbar arteries, which simplifies the experimental procedure. Furthermore, the arrangement of the spinal radicular arteries above the ARM were analogous to those present at a higher level in primates [16], although the porcine radicular arteries are smaller and more numerous. Of interest, in the 9 porcine spinal cord specimens we studied, the anterior spinal artery, although small when compared with that of primates, was continuous from the midthorax to the sacrum. An important difference, which has been noted before [21], is that the spinal cord in the pig terminates at a lower vertebral level than in primates, and therefore, a laminectomy is the preferable route for introducing a catheter rather than through a percutaneous lumbar puncture.

We have previously reported our success in protecting the spinal cord from ischemia during aortic cross-clamping in both nonhuman primates [7] and humans [1, 14, 15] by the use of intrathecal administration of papaverine. However, in the nonhuman primate experiments, the segmental intercostal or lumbar arteries were rarely sacrificed, and thus the spinal cord blood supply was reestablished when the aorta was unclamped. The advantage of intrathecal administration of papaverine combined with this new HICI technique is that, apart from possibly increasing its sensitivity by dilating the radicular arteries ($p < 0.025$; see Table 1) and anterior spinal artery ($p < 0.05$; see Table 1), the papaverine may allow more time for the reanastomosis of critical segmental arteries. The additive protection of both these evolving techniques and the use of distal aortic perfusion under certain circumstances [10, 22] requires further research but may reduce the incidence of paraplegia after aortic operations. The role of spinal and cortical motor evoked potentials requires further evaluation; however, our [7, 8] earlier results with cortical and spinal stimulation were accurate and encouraging reviews have been published [23].

In conclusion, this study has addressed the problem of localizing segmental arteries that supply the spinal cord so that they can be reattached during aortic replacement, thus reestablishing the blood supply to the spinal cord. We have shown that in the porcine model, the radicular arteries supplying the spinal cord can be accurately and rapidly localized using HICIs and that the entire spinal cord from the lower thoracic segments to further caudally can be perfused by these radicular arteries. Currently we are studying the safety of this method and the influence of preservation or occlusion or perfusion of HICl-localized segmental arteries, combined with motor evoked potentials, on the incidence of postoperative paraplegia in the porcine model. With further refinement and testing for safety, we have used this technique, including spinal motor evoked potentials, in humans to rapidly identify segmental arteries that supply the spinal cord, which then have been reattached during aortic replacement to preserve spinal cord function.

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DISCUSSION

DR AHMAD RAJAII-KHORASANI (Boston, MA): The importance of localization of the spinal cord blood supply becomes more evident when I discuss my technique for aortic operations. First, however, let me point out that both Dr Cooley and Dr Crawford have mentioned that whether you reimplant the intercostal arteries or not, the chance of paraplegia is the same. This bitter experience is, in a way, predictable considering the sensitivity of the central nervous system tissues to ischemia. In other words, if these vessels are critical to the spinal cord blood supply, by the time they are reimplanted, the damage has already been done.

The technique that I have used since 1986 involves use of a high-flow shunt with a branched sidearm. (The sidearm can also originate from the inflow line of the bypass machine.) This system makes possible adequate distal perfusion as well as perfusion of the excluded intercostals. Adherence to the following principles is essential: (1) maintain adequate distal circulation; (2) minimize the number of excluded intercostals by proper choice of clamp site and avoid clamp injury to the neighboring intercostals; (3) reperfuse the excluded intercostals immediately after aortotomy; and (4) reimplant these intercostals to the side of the aortic graft without interruption of their blood flow.

This technique will minimize the chance of spinal cord ischemia, and with localization of the spinal cord blood supply, the technique can be simplified tremendously. Further investigation, especially with animals that have variation in spinal cord blood supply, is needed.

DR SAFEH ATTAR (Baltimore, MD): Dr Svensson, in your experimental results, you indicated that drainage of the cerebrospinal fluid was not helpful in the prevention of paraplegia. Now, you present a series in which you used papaverine and cerebrospinal fluid drainage combined and cerebrospinal fluid drainage by itself. Did you use papaverine by itself to see its effect?

Also, in a personal communication, Larry Hollier at the Ochsner Clinic indicated that he had operated on 50 patients with thoracoabdominal aortic aneurysms using cerebrospinal fluid drainage and reimplantation of all the involved intercostal and lumbar arteries. There were only 2 cases of paresis, which improved after onset by drainage of the cerebrospinal fluid. How do you reconcile this? This is a practical way of preventing paraplegia that has been proved clinically.

DR SVENSSON: Dr Rajaii-Khorasani, with reference to reanastomosis of the intercostals by Dr Cooley and Dr Crawford, I must state on Dr Crawford's behalf that he has stressed the importance of reanastomosis of the intercostals in two reports, one in 1979 in *Surgical Clinics of North America* and one in 1986 in *The Journal of Vascular Surgery.*

Concerning perfusion of the spinal cord, the matter does not appear to be resolved in human studies. My colleagues and I in Houston do not believe that distal aortic perfusion in humans has much advantage in operations for the medial degeneration type of thoracoabdominal aneurysm, partly for anatomical reasons that I have explained.

As for your operative technique, I believe that it is similar to the original technique of repairing thoracoabdominal aneurysms. The reason that technique was abandoned was the high mortality rate. Currently in our prospective randomized study of cerebrospinal fluid drainage, we have a 30-day mortality rate of 3%. The effort to prevent paraplegia should not be accomplished at the expense of an increased mortality rate.

Dr Attar, in answer to your questions about intrathecal papaverine, we did not use intrathecal papaverine alone in the animal studies because we had either done a laminectomy or inserted a catheter; therefore, cerebrospinal fluid was lost. Thus, papaverine was used with cerebrospinal fluid drainage. In the study just presented, we did not, however, evaluate the effect of these measures or HICI localization on postoperative paraplegia. This will be the subject of further research.

With reference to the results from the Ochsner Clinic under the auspices of Dr Hollier, McCullough and associates reported that many of those patients had Crawford type III or IV aneurysms, which have an expected combined rate of paraplegia and paresis of only 3.2% and 2.1%, respectively. Thus, they were low-risk patients.

We are currently testing the effect of cerebrospinal fluid drainage alone in a prospective randomized study in high-risk patients with a combined paraplegia and paresis rate in the control group running at about 25% to 30%. To date, we have found no significant advantage with cerebrospinal fluid drainage alone. We will report these results when we have completed the study.

Cerebrospinal fluid drainage alone was used in animal experiments more than 20 years ago. Recently, there has been a resurgence of research to try to determine whether such drainage is advantageous. Nonetheless, Dr Cooley and Dr Blaisdell have both commented that cerebrospinal fluid drainage alone did not prevent paraplegia or paresis in experiments they reported in 1962. Later in 1965, Dr Killen and colleagues repeated the studies and found no protective effect of cerebrospinal fluid drainage from the cisterna magna in dogs. This is the technique that is still used today in canine experiments. There appears to be little advantage in using cerebrospinal fluid drainage alone without other adjuncts.

Effect of Antireflux Operation on Barrett's Mucosa

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Regression of Barrett's epithelium after antireflux operations remains a controversial topic. We evaluated the effect of antireflux procedures in patients with Barrett's esophagus on the regression of columnar epithelium and dysplasia and its potential protective effect on the subsequent development of carcinoma. Of the 241 patients with Barrett's esophagus treated at the Lahey Clinic from 1973 to 1989, 37 patients underwent an antireflux operation. Regression was defined as histological evidence of regenerating squamous mucosa that completely or partially replaced the columnar epithelium. Improvement in lower esophageal sphincter pressure to 12 mm Hg or greater occurred in 19 of 26 patients (73%) who had perioperative manometry. Symptomatic relief of esophagitis occurred in 34 of 37 patients (92%). Four patients had partial regression with regenerating squamous mucosa juxtaposed with areas of columnar epithelium. Carcinoma developed in 3 of 37 patients (8.1%). One patient had recurrence of severe symptoms of reflux esophagitis before development of carcinoma. Patients with Barrett's esophagus who have undergone a successful antireflux operation with symptomatic relief and evidence of improvement in lower esophageal sphincter pressures rarely show regression of Barrett's mucosa and may still be at risk for development of carcinoma. Therefore, the indications for antireflux operation in Barrett's esophagus should remain the same as for other patients with gastroesophageal reflux, but yearly endoscopic and histological surveillance should be continued postoperatively.

(Ann Thorac Surg 1990;49:537–42)

Barrett's esophagus (BE) is one of the complications of long-standing gastroesophageal reflux [1–7]. Continued mucosal acid-peptic injury results in destruction of the normal squamous mucosal lining and subsequent replacement with columnar epithelium, although the precise origin of this epithelium remains controversial [8–10]. The premalignant nature of this abnormal epithelium is well known [11–16]. The development of Barrett's epithelium and its transition to dysplasia and ultimately in some patients to carcinoma may be related to continued peptic reflux injury. The question remains, however, whether control of reflux in these patients will prevent development of dysplasia and reduce the incidence of carcinoma. In an effort to resolve this uncertainty, records of all Lahey Clinic patients with BE who underwent antireflux procedures from 1973 to 1989 were reviewed. We wished to determine whether control of acid-peptic reflux by successful antireflux operation results in regression of Barrett's epithelium and dysplasia and whether it reduces the likelihood of subsequent development of carcinoma.

For editorial comment see page 523.

Material and Methods

Definitions

For this study, BE was defined as one in which the columnar mucosal lining circumferentially involved 3 cm or more of the distal tubular esophagus identified endoscopically. Regression of Barrett's epithelium was determined histologically by evidence of regenerating squamous mucosa replacing the columnar epithelium accompanied by a decrease of 3 cm or greater in the total length of BE. This length was calculated as the distance from the squamocolumnar junction to the caudal end of the tubular esophagus. Carcinoma arising in BE was defined as adenocarcinoma originating cephalad to the junction of the tubular esophagus and stomach and associated with the adjacent characteristic columnar mucosa.

Patient Population

Of 241 patients with BE treated at the Lahey Clinic Medical Center from 1973 to 1989, 65 had carcinoma and BE. The remaining 176 patients were followed with yearly endoscopic surveillance and biopsy studies. Of these patients, 37 underwent a total of 44 antireflux procedures and are the subject of this review, which includes 20 patients reported previously [17]. Patients varied in age from 19 to 68 years (median age, 52 years). Of these patients, 28 (75%) were men and 9 were women. The indications and types of antireflux procedures performed are shown in Tables 1 and 2. The majority of procedures (84%) were Nissen fundoplications. A Collis gastroplasty was added in 34% of patients. Four patients had two antireflux procedures, and 1 patient had three. One patient had esophagogastrectomy with resection of the

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Table 1. Indications for Antireflux Procedure in Patients with Barrett's Esophagus

Indication	No. of Patients
Severe reflux symptoms	23
Stricture with dysphagia	9
Nonhealing ulcer	4
Bleeding	1

Table 3. Initial Symptoms

Symptom	No. of Patients
Heartburn	32
Regurgitation	17
Dysphagia	17
Bleeding or anemia	7
Weight loss	3

stricture, antrectomy, and Roux-en-Y jejunojejunostomy for refractory reflux and stricture.

The initial symptoms in this group of patients are shown in Table 3. Strictures were present in 9 of 37 patients (24%), and 4 of 37 patients (11%) had Barrett's ulcers. Symptomatic status after operation was assessed by chart review as well as by questionnaire. Patients reported complete relief of reflux symptoms as excellent, marked improvement in symptoms as good, or no change or worsening of symptoms as poor.

Esophagoscopy

All patients had yearly endoscopic evaluation with multiple biopsies at 2-cm to 3-cm intervals in representative quadrants. Patients in whom dysplasia developed underwent repeat endoscopy in 1 month and at 3 months provided the dysplasia resolved. Endoscopic follow-up studies ranged from 4 months to 12.5 years (median, 4.2 years).

Esophageal Manometry and Standard Acid-Reflux Testing

Of the 37 patients, 31 (84%) underwent preoperative esophageal manometry, and 17 patients had standard acid-reflux testing. Of these patients, 26 also had early postoperative manometric studies and 13 had standard acid-reflux testing. Lower esophageal sphincter (LES) pressure was considered normal at 12 mm Hg or greater, and results of standard acid-reflux testing were considered normal at a pH of 4.0 or greater.

Pathological Findings

Dysplasia was graded according to the revised criteria of Riddell and colleagues [18–20]. The three categories were negative for dysplasia, mild dysplasia, and severe dysplasia. These groupings were based on nuclear abnormalities, with mild dysplasia characterized by nuclei largely confined to the basal portion of the cells and severe dysplasia characterized by nuclei situated in the upper pole of the cell. In addition to polarity, other nuclear characteristics of dysplasia were hyperchromatism, enlargement, stratification, pleomorphism, and abnormal mitosis. Nucleolar changes diagnostic of dysplasia included enlargement and multiplicity.

Table 2. Antireflux Procedures Performed

Procedure	No.[a]
Nissen fundoplication	22 (50)
Collis gastroplasty and Nissen fundoplication	15 (34)
Belsey procedure	2 (5)
Collis gastroplasty and Belsey procedure	4 (9)
Acid suppression, alkaline diversion	1 (2)

[a] Numbers in parentheses are percentages.

Results

Thirty-four of 37 patients (92%) had good to excellent relief of symptoms of reflux esophagitis. Long-term postoperative follow-up time in 26 patients ranged from 2 to 12.5 years (median, 5 years) and showed continued good to excellent symptomatic results in 21 of 26 patients (81%).

Of 9 patients with dysphagia caused by stricture in whom conservative management with dilation and medical therapy had previously failed, 7 had complete relief of dysphagia and resolution of stricture after the antireflux operation. All patients with Barrett's ulcers measuring 1 to 1.5 cm in whom the ulcer failed to heal with medical therapy experienced resolution after antireflux operation.

No evidence of active inflammation or ongoing esophagitis was endoscopically evident in 30 of 37 patients in their first year after operation. Long-term endoscopic follow-up in 20 patients showed no recurrence of esophagitis. In 4 patients, however, evidence of mild esophagitis was observed endoscopically, and 3 patients had recurrence of severe esophagitis.

Antireflux operation resulted in normalization of LES pressures in 19 of 26 patients (73%) who had both preoperative and postoperative manometric studies. Five more patients (19%) had improvement in LES pressures to at least 10 mm Hg. Changes in LES pressures in these 26 patients, which were statistically significant ($p < 0.0001$), are shown in Fig 1. Postoperative standard acid-reflux testing showed normal results in 8 of 13 patients (62%), but results remained positive in 5 patients.

None of our patients had complete regression of Barrett's mucosa. Four patients (11%) showed endoscopic evidence of partial regression, with regenerating squamous mucosa infiltrating areas of columnar mucosa accompanied by a decrease of 3 cm or greater in the total length of BE. Three of these patients had both preoperative and postoperative manometry, which demonstrated postoperative LES pressures of at least 15 mm Hg.

Likewise, development of dysplasia did not appear to be altered appreciably by antireflux operation. Despite symptomatic and endoscopic improvement in reflux esophagitis after successful antireflux procedures, mild dysplasia later developed in 4 patients. In 2 of these

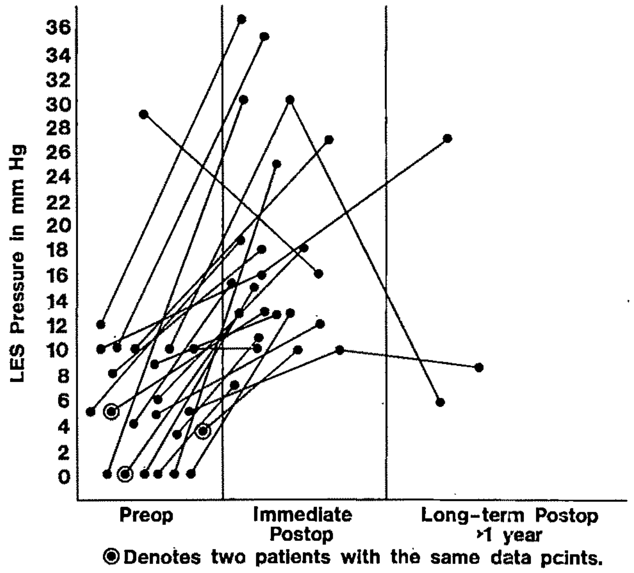

Fig 1. Preoperative and postoperative lower esophageal sphincter (LES) pressures.

patients, mild dysplasia progressed to the severe form and to carcinoma at 1 and 10 years after operation. In 1 patient, severe dysplasia developed after operation and progressed to carcinoma without first showing mild dysplastic changes. In fact, only 1 patient in our series had mild dysplasia diagnosed preoperatively. At 2 years after a Collis-Nissen fundoplication, this patient has no further evidence of dysplasia.

The 3 patients in whom carcinoma developed all had manometric evidence of an adequate antireflux procedure with improvements in LES pressures from 0 to 10, 9 to 15, and 10 to 30 mm Hg. All patients had endoscopic resolution of esophagitis and good to excellent relief of symptoms. Only 1 patient had endoscopic recurrence of esophagitis with stricture accompanied by a decrease in LES pressure to 6 mm Hg before development of severe dysplasia and carcinoma. The other 2 patients had no endoscopic recurrence of esophagitis. By the time dysplasia and carcinoma developed, 1 patient had recurrence of severe heartburn and dysphagia, 1 patient had mild heartburn controlled medically, and the third patient was asymptomatic.

The incidence of carcinoma in patients with BE who had antireflux procedures was 1 in 48 patient-years of follow-up. This incidence is higher than the incidence in our entire population of patients with BE (1 in 99 patient-years of follow-up), although it is not statistically significant ($p = 0.34$) by Miettinen's modification of the Fisher exact test (Williamson WA, et al, unpublished observations, Aug 1989).

Comment

The question of regression of Barrett's epithelium after an antireflux operation cannot be answered from a review of the literature. In five studies [21–25] involving 49 patients, 6 patients demonstrated partial regression and 3 showed complete regression. Other studies [6, 7, 26–28] have failed to demonstrate any evidence of regression.

A lack of uniformity in defining regression complicates this issue. Defining regression as an arbitrary change in the level of the squamocolumnar junction in relationship to the incisors is incorrect because antireflux operations inevitably reposition the esophagogastric junction and thus shift the squamocolumnar junction distally. This event does not represent regression.

Skinner and associates [21] were first to describe the entity of regenerating squamous epithelium overlying residual columnar mucosa. They noted a change in the appearance of the columnar mucosa from red to pink, which contrasted with the pale coloration of the adjacent normal squamous mucosa in 2 of their patients. When they performed a biopsy on this area with a large cup biopsy forceps, they found that the squamous epithelial lining was overgrowing the intact deeper columnar mucosal layer. Whether this represents the early stages of regression, which later progresses to atrophy of the deeper columnar layer, or whether this deeper layer persists indefinitely along with its potential for malignant degeneration is speculative. None of our patients with partial regression had underlying columnar mucosa. The biopsies may not have been deep enough to demonstrate this feature, however.

The technique and number of biopsies have been controversial issues in assessing regression of Barrett's epithelium. For example, in 2 of the 4 patients with apparent regression reported by Brand and co-workers [25], only one preoperative biopsy site in the esophagus was sampled. In addition, these researchers used a blind suction biopsy after operation rather than an endoscopically directed biopsy technique. They did, however, use the absolute length of columnar mucosa from the distal tubular esophagus to the squamocolumnar junction to document regression. On the average, our patients had four biopsies. Recently, our surveillance involves more extensive biopsies and now averages five to six per patient.

Documentation of an adequate antireflux procedure has been another criticism of some studies [29–31]. Reliance on symptomatic criteria alone may be particularly misleading in judging the adequacy of antireflux procedures in patients with BE because the columnar epithelium is less sensitive to pain [32]. Assessment of LES pressure with esophageal manometry, standard acid-reflux testing, and resolution of esophagitis by endoscopy are more accurate ways of judging the adequacy of antireflux procedures. Of our 26 patients, 19 (73%) had adequate antireflux procedures using manometric criteria. Five more patients had appreciable improvement in LES pressures to at least 10 mm Hg. To our knowledge, this report and the report by Skinner and Booth [33] are the only two that record preoperative and postoperative manometry or acid-reflux testing in this population. One criticism of our series would be the lack of long-term manometric data. Only 4 patients had manometry 1 to 8 years after their early postoperative studies. Long-term endoscopic surveillance was performed, however. These data demonstrated early postoperative resolution of esophagitis in 30 of 37 patients, and this result persisted in 20 of 27 patients for a median follow-up of 5 years (range, 2 to 12.5 years).

Manometry and endoscopy, however, are at best indirect assessments of reflux. Although patients with abnormal reflux have LES pressures that are lower than normal, much overlap occurs among patients with refluxing and nonrefluxing esophagitis over a wide range of LES pressures. Acid-reflux testing is a more sensitive indicator of abnormal reflux [33]. The most sensitive test for detection of acid reflux is 24-hour pH monitoring, which was not performed in any of our patients. Nonetheless, we believe that manometric evidence of improvement in LES pressures, supported by acid-reflux testing in some patients, and endoscopic resolution of esophagitis constitute adequate objective evidence of control of reflux in all but a few of our patients, lending more validity to our conclusion regarding the ultimate fate of Barrett's mucosa after antireflux operation.

Whether surgical control of reflux had any impact on the postoperative development or resolution of dysplasia in our patients is uncertain because sampling error can lead to false-negative biopsies; however, 1 of the 3 patients in whom carcinoma developed clearly demonstrated a pattern of initial postoperative relief of heartburn with subsequent recurrence of symptoms 3 years later. Coincident with this finding, endoscopy revealed a progression from severe dysplasia to carcinoma. Lower esophageal sphincter pressures increased from 10 to 30 mm Hg initially and then decreased to 6 mm Hg, and results of standard acid-reflux testing changed from positive to negative to positive again over the same 3-year interval. The other 2 patients in whom carcinoma developed had good relief of heartburn and regurgitation and resolution of esophagitis endoscopically, yet the biopsies in both patients showed progression from mild dysplasia to severe dysplasia to carcinoma in situ within 1 year and 10 years after operation.

Antireflux procedures may provide symptomatic relief to patients with BE and may promote healing of esophagitis, strictures, and Barrett's ulcers. Actual regression of Barrett's epithelium occurs infrequently (4 of 37 patients in our series), however, and is rarely complete. Even when patients with BE undergo a successful antireflux procedure, documented by objective criteria, they remain at risk for the development of dysplasia and carcinoma.

We conclude, therefore, that the indications for antireflux operations in patients with BE should be the same as for other patients with gastroesophageal reflux and that frequent endoscopic surveillance should be mandatory for all patients with BE even after an antireflux operation.

We thank Gerald J. Heatley, MS, of the Sias Surgical Research Unit for statistical assistance in the preparation of this report.

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DISCUSSION

DR GORDON F. MURRAY (Morgantown, WV): Dr Williamson and colleagues, in this endoscopically well documented series of 37 patients undergoing antireflux procedures, have provided compelling evidence that the present operative treatment of patients with BE is in principle analogous to that used in patients with reflux disease.

Similarly, evidence prevails that long-term treatment with H_2-receptor antagonists and antacids has a beneficial effect with regard to symptomatology and endoscopic signs of esophagitis and ulceration but does not result in a regression of the metaplastic epithelium.

A conclusion that the columnar-lined esophagus and its association with the development of adenocarcinoma of the esophagus defies surgical intervention and prevention should be resisted.

It does appear that the abnormal Barrett's mucosa is exquisitely sensitive to pH. If there is to be regression of the precancerous columnar epithelium, nearly complete removal of the acid exposure may be required. Long-term manometric data were provided for only 4 patients in this study. Postoperative standard acid-reflux test results were normal in only 8 of 13 patients and remained positive in 5 patients. The more sensitive indicator of abnormal reflux—24-hour pH monitoring—was not performed in any of the present patients. Therefore, even this fine effort leaves doubt as to the efficacy of acid-reflux elimination.

Accordingly, the clinical influence of the drug Omeprazole, which produces proton pump paralysis, is of great interest, particularly in proposed clinical trials directed at patients with chronic reflux disease characterized by columnar metaplasia. Omeprazole is a member of a new class of gastric antisecretory agents that specifically inhibit the enzyme hydrogen-potassium adenosine triphosphatase in the parietal cell, which is the final step in the formation of hydrochloric acid. It is a highly effective inhibitor of gastric acid secretion and produces more complete and long-lasting suppression of basal and stimulated gastric acid secretion than currently available H_2-receptor antagonists.

To be noted is that 4 patients in the current series did have regression with regenerating squamous mucosa. Also, I am struck that at least 1 patient in this series had dysplasia diagnosed preoperatively, and 2 years after a Collis-Nissen fundoplication had no further evidence of dysplasia.

Dr Williamson, is there any further correlation between the esophageal lengthening procedure and favorable outcome in these patients? An alliance between pharmacology and surgical innovation may yet carry the day.

DR DAVID B. SKINNER (New York, NY): This would be an important study if in fact the control of reflux were well documented in the patients that progressed to carcinoma, but I have the same line of questions that Dr Murray has raised.

You indicated that 24 of your 37 patients had an improvement in manometry, but apparently some did not, and we do not know the results of the pH testing. Were the patients that progressed to carcinoma in fact documented to have had a successful antireflux repair by pH testing and manometry?

Related to that, were the progressions to carcinoma in patients with a Collis gastroplasty or were they in patients with a straightforward Nissen fundoplication? We have heard from Dr Pearson at this meeting in previous years that some of the Collis gastroplasties do acquire adenocarcinoma at a later time. Again, was the carcinoma in the Barrett's segment or in the Collis segment if that was the case?

Finally, the regeneration of squamous mucosa may not occur in a progressively downward fashion from the squamocolumnar junction. What we have seen in about a quarter of our patients is a spreading out of islands of squamous epithelium in areas of the esophagus that were previously lined with Barrett's but without a definitive downward progression of the mucosal junction. Have you seen that phenomenon?

DR JERRY C. ROSENBERG (Detroit, MI): Like Dr Murray and Dr Skinner, I am unhappy that a fundoplication does not reverse the metaplastic epithelium of a BE. However, unlike them, I think Williamson and associates have convincing data that correcting the reflux does not produce regression of the columnar cells to squamous cells. I would therefore like to know whether, short of doing an esophagectomy, it is worthwhile to ablate the abnormal mucosa at the time that one corrects the reflux. Has that ever been tried? Does that have any merit?

DR JOHN R. BENFIELD (Sacramento, CA): This is an important paper. It is important, however, to keep in mind that the control for this series of patients is the patients who are not operated on. The data that have been presented indicate the possibility that the antireflux procedure may just be an interlude, in some instances, in the progression of the disease to carcinoma. Dr Williamson, do you have any information as to the regression rate of dysplastic changes in the esophagus among patients you have followed during nonoperative treatment?

DR WILLIAMSON: Concerning the issue of regression of dysplasia in the nonoperative group, mild dysplasia developed in 20 of 176 patients while they were under endoscopic surveillance. Fifteen of these patients had not undergone antireflux procedures and were treated medically. None of these fifteen patients progressed to carcinoma. There were 2 patients in the medically treated group, however, in whom severe dysplasia and carcinoma developed. This incidence was not significantly different from the incidence of carcinoma in patients who underwent antireflux procedures. One of the difficulties in trying to assess the regression of dysplasia in these patients is that sampling error can lead to false-negative biopsies.

We recognize the lack of 24-hour pH monitoring in our patients as a shortcoming of this study. However, with this test one may have up to 49 episodes of acid reflux during a 24-hour period and still be considered normal according to the criteria established by Skinner and DeMeester. What is not clear, however, is how many episodes of reflux per day are required to stimulate the progression to dysplasia and carcinoma in patients with BE. Even 24-hour pH monitoring may not be sensitive enough to evaluate the effectiveness of antireflux operation in these patients.

Only 1 of the 3 patients in whom carcinoma developed after an antireflux operation had a Collis gastroplasty.

The question posed by Dr Murray about whether the complete removal of acid is necessary in these patients is a good one. In a paper presented last week at the International Society for Diseases of the Esophagus, Dr Fekete and his colleagues reported on 25 patients with BE who had an antrectomy, vagotomy, and Roux-en-Y gastrojejunostomy as their antireflux procedure. Five of these patients had partial regression of their Barrett's mucosa and one had complete regression at 6 years postoperatively. Perhaps the complete removal of acid is necessary to see regression, or perhaps the removal of alkaline reflux is equally important in protecting these patients.

Relationship Between Total Mechanical Energy and Oxygen Consumption in the Stunned Myocardium

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We studied the relationship between left ventricular oxygen consumption ($LVVo_2$) and total ventricular mechanical energy production as determined by calculation of the systolic pressure–volume area (P-VA) before and after 25 minutes of warm ischemia in 7 sheep. We compared the relationship between $LVVo_2$ and P-VA with the relationships between $LVVo_2$ and stroke work and between $LVVo_2$ and the systolic stress integral. Using the methods presented, P-VA can be measured in vivo (n = 123) in both preischemic and postischemic hearts. Ischemia increases the slopes of the relationship between $LVVo_2$ and P-VA and between stroke work and the systolic stress integral, and reduces the oxygen utilization efficiency of stroke work to less than 2%. Coefficients of determination for the relationship between $LVVo_2$ and P-VA are, in general, higher than those between $LVVo_2$ and either stroke work or the systolic stress integral. We conclude that systolic P-VA can be measured in vivo using recently developed methods and that it is applicable to postischemic "stunned" hearts. Because P-VA and $LVVo_2$ can be converted into identical energy units, calculation of P-VA permits calculation of myocardial oxygen utilization efficiency.

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Study of the relationship between mechanical energy produced by the heart and oxygen consumed necessitates quantitation of the many independent variables that alter mechanical energy generation. To simplify predictions of myocardial oxygen consumption, several indices of mechanical energy production have been developed [1–3]. One of the most popular is the systolic pressure–volume area (P-VA) developed by Suga and Sagawa [4]. These investigators have shown that for a given contractile state, P-VA linearly correlates with myocardial oxygen consumption over a wide range of physiological conditions in the excised perfused canine heart, estimates the ratio between external (mechanical) work and "potential energy," and is an energy term that can be used to calculate myocardial oxygen efficiency. However, P-VA has not been studied in poorly contractile hearts in vivo.

For editorial comment see page 525.

Recently we [5] found that left ventricular oxygen consumption ($LVVo_2$) significantly increases after reversible global ischemia in sheep and linearly correlates with the integral of left ventricular systolic stress (SSI), which is a measure of ventricular external work. We tested the hypothesis that reversible global ischemia increases myocardial oxygen consumption in relation to total ventricular energy production as measured by P-VA and reduces oxygen utilization efficiency.

Material and Methods

Twenty-two healthy Dorset sheep (mean weight 35.3 ± 5.4 kg) were studied in compliance with the "Guide for the Care and Use of Laboratory Animals" published by the National Institutes of Health (NIH Publication No. 85-23, revised 1985). Complete data before and after 25 minutes of global myocardial ischemia were obtained in 7 sheep, and they are the subject of this report.

Techniques

Details of the methodology and some data from 5 sheep are included in a previous report [5]. Briefly, all animals had an initial sterile left thoracotomy for placement of ultrasonic transit-time flow probes (Transonic Inc, Ithaca, NY) around the left main coronary artery (4 to 6 mm) and around the ascending aorta (16 to 20 mm). The hemiazygos vein was ligated, and a 6-mm hemispherical crystal (3 mHz) (model H25-C; Sonotek Inc, Del Mar, CA) was sutured to the base of the heart between the aorta and the main pulmonary artery.

One to 2 weeks later, the heart was exposed through a midline sternotomy incision. One apical and three additional pairs of sonomicrometry crystals were placed on the left ventricle to measure two orthogonal equatorial axes, the longitudinal axis and wall thickness. Micromanometer pressure transducers (model SPC-350; Millar Instruments, Inc, Houston, TX) were placed in the aortic arch, superior vena cava, and left ventricle. A fiberoptic oxygen-saturation catheter (model P7110; Oximetrix Co, Mountain View, CA) was placed into the coronary sinus. Small

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intraaortic balloons (Datascope, Inc, Oakland, NJ) were placed through the right carotid and left femoral arteries and positioned in the ascending and descending aorta. The electrocardiogram was monitored continuously.

End-systolic elastance (Ees) was measured in each preischemic heart by the aortic occlusive single-beat method [6]. Left ventricular pressure–volume relationships and simultaneous left ventricular coronary artery blood flow and coronary sinus oxygen saturation measurements were made during 20-second intervals at various afterloads as previously described [5]. After afterload was changed by partial inflation of the descending thoracic aortic balloon, a 90-second interval to achieve steady-state conditions preceded each 20-second period of data collection. Each measurement at increased afterload was alternated with a measurement without inflation of the descending thoracic aortic balloon. Hemoglobin and arterial oxygen tension were measured intermittently during data collection periods.

Sheep were given heparin sodium and were cannulated for cardiopulmonary bypass after five to 13 separate 20-second measurements in preischemic hearts. Cardiopulmonary bypass was started at 37°C; a venting catheter was placed in the apex of the left ventricle, and the aorta was cross-clamped for 25 minutes (standard deviation, 0.3 minute). After release of the aortic clamp, the heart was defibrillated and reperfused on bypass with the apical vent open during infusions of both epinephrine (0.35 μg/kg/min) and lidocaine hydrochloride (1 mg/min). All drugs were stopped at least ten minutes before determination of postischemic Ees, $LVVo_2$, and left ventricular volume and pressure as previously described [5]. Postischemic measurements were made 84 minutes (standard deviation, 12.3 minutes) after release of the aortic clamp.

Calculations

Transit-time coronary artery blood flow measurements and measurements of left ventricular volume by sonomicrometric measurements of three orthogonal axes using an ellipsoid model have been previously validated [5, 7]. The relevant calculations for $LVVo_2$, left ventricular volume at various phases of the cardiac cycle, and SSI have also been published [5]. The $LVVo_2$ data were normalized to a 100-g left ventricle, which was trimmed and weighed immediately after death, and to each heart beat. In sheep, the left main coronary artery exclusively supplies the left ventricle [8]; therefore, $LVVo_2$ can be calculated.

The Ees was determined from four single-beat volume–pressure loops recorded before and after ischemia using the aortic root impedance balloon [6]. The Ees was generated from the E_{max} (the point of maximum pressure decided by volume) of each loop using a shooting-point numerical iterative method [9]. Left ventricular volume at zero pressure (V_0) was determined by extrapolation of Ees. The V_0 and Ees were determined in each preischemic and postischemic heart before each measurement of $LVVo_2$ and ventricular volume and pressure.

Systolic pressure–volume area is defined as the area enclosed by the systolic segment of the pressure–volume loop and two lines drawn from V_0 to E_{max} and to the end-diastolic pressure–volume curve. This area, in units of millimeters of mercury × milliliters, is determined by computerized planimetry and subdivided into stroke or external work, defined as the area within the entire pressure–volume loop, and into internal work or end-systolic potential energy, defined as the triangular area between V_0 and isovolumic diastole of the pressure–volume loop. The P-VA and external work are converted to joules by multiplying each area (in units of millimeters of mercury × milliliters) by 1.33×10^{-4} [3]. Oxygen utilization efficiency (O_2UE) was calculated using the physiological conversion factor for normal hearts of 1 mL of oxygen approximately equals 20 J [1–3]: O_2UE (%) = P-VA (J) × $10^2/LVVo_2 \times 20$ (J).

Commercially available software (BBN Software RS/1,

Table 1. Hemodynamic Data[a]

Sheep No.	Ischemic State	No. of Measurements	HR (beats/min)	CO (L/min)	LVP (mm Hg)	Ees (mm Hg/mL)	dP/dt (mm Hg/s)
1	Before	5	167 (2)	1.35 (0.2)	83.6 (1.2)	2.71 (0.3)	1,844 (142)
1	After	7	123 (4)	0.9 (0.1)	50.2 (2.4)	1.10 (0.4)	755 (82)
2	Before	7	106 (2)	1.1 (0.2)	45.2 (2.3)	1.17 (0.2)	633 (37)
2	After	5	79 (3)	0.7 (0.2)	47.8 (2.7)	1.06 (0.2)	737 (58)
3	Before	13	101 (2)	1.63 (0.1)	46.4 (2.8)	1.33 (0.2)	716 ((34)
3	After	6	94 (2)	0.5 (0.1)	45.9 (1.8)	1.42 (0.3)	668 (66)
4	Before	5	111 (3)	0.6 (0.1)	77.6 (2.9)	3.67 (0.3)	1,467 (121)
4	After	9	88 (4)	0.3 (0.1)	48.7 (1.9)	11.58 (0.1)	890 (86)
5	Before	7	131 (4)	2.1 (0.2)	107.6 (3.3)	5.24 (0.2)	2,149 (55)
5	After	8	104 (5)	0.2 (0.1)	34 (4.1)	1.52 (0.3)	571 (76)
6	Before	11	131 (3)	2.1 (0.2)	75.3 (1.2)	6.33 (0.4)	1,281 (112)
6	After	6	81 (4)	1.1 (0.1)	47.2 (2.5)	2.99 (0.2)	705 (89)
7	Before	7	178 (4)	1.2 (0.1)	70.6 (2.2)	4.64 (0.2)	1,993 (146)
7	After	11	121 (4)	0.9 (0.1)	49.3 (1.1)	2.57 (0.3)	931 (63)

[a] Data are shown as the mean of measurements made at different afterloads with the standard error in parentheses.

CO = cardiac output; dP/dt = maximum rate of rise of left ventricular pressure; Ees = end-systolic elastance; HR = heart rate; LVP = left ventricular pressure.

Table 2. Mean Oxygen Consumption and Indices of Energy Production[a]

Sheep No.	Ischemic State	No. of Measurements	$LVVo_2$ (μL/100 g/beat)	$LVVo_2$ (J/beat)	P-VA (J)	EW (J)	SSI (mm Hg/s)
1	Before	5	106 (3)	2,112 (60)	124 (9)	115 (7)	9.16 (0.45)
1	After	7	88 (2)	1,764 (26)	72 (6)	58 (4)	8.19 (0.36)
2	Before	7	50 (3)	1,000 (62)	30 (9)	25 (8)	9.93 (0.37)
2	After	5	55 (3)	1,106 (52)	67 (8)	41 (6)	8.25 (0.52)
3	Before	13	67 (4)	1,348 (77)	73 (11)	52 (10)	19.46 (0.86)
3	After	6	109 (3)	2,170 (66)	32 (4)	8 (2)	16.50 (0.951)
4	Before	5	63 (2)	1,254 (39)	85 (4)	62 (8)	13.03 (0.73)
4	After	9	72 (2)	1,432 (33)	65 (6)	19 (3)	3.15 (0.52)
5	Before	7	46 (1)	920 (28)	254 (12)	233 (11)	22.54 (1.06)
5	After	8	32 (1)	644 (21)	43 (3)	12 (3)	12.39 (0.75)
6	Before	11	41 (2)	820 (32)	46 (7)	35 (6)	9.43 (0.92)
6	After	6	119 (1)	2,378 (28)	27 (5)	25 (4)	6.39 (0.53)
7	Before	7	40 (2)	796 (41)	26 (4)	18 (3)	9.14 (0.66)
7	After	11	42 (1)	834 (12)	21 (3)	11 (2)	7.97 (0.73)

[a] Data are shown as the mean of measurements made at different afterloads with the standard error in parentheses.

EW = external or stroke work; $LVVo_2$ = left ventricular oxygen consumption; P-VA = systolic pressure–volume area; SSI = systolic stress integral.

Systat) was used for statistical analysis. Numerical data were compared after tests for normality by Student's unpaired *t*-test. Linear regression equations were calculated by the method of least squares and compared after Fisher's Z transformation.

Results

Averaged basic hemodynamic data from each sheep before and after ischemia are presented in Table 1. Because the data were obtained at different afterloads, these data provide only background information. The surgical intervention required for instrumentation caused low cardiac output in most sheep before ischemia. Ischemia consistently reduced heart rate and usually reduced peak left ventricular pressure and both measures of contractile state, Ees and rate of rise of left ventricular pressure.

Table 2 presents averaged data relating to $LVVo_2$ and the indices of left ventricular energy production at different afterloads. As expected, ischemia reduced the indices of left ventricular energy production, but the change in $LVVo_2$ was inconstant.

Three indices of left ventricular energy production are compared in Figure 1. The correlations between SSI [7, 10] and both stroke work (external work) and P-VA were similar. At lower levels of wall stress, correlations were good (see Figure 1); at higher levels of stress, the data scattered and confidence limits diverged.

Because the relationship between $LVVo_2$ and indices of left ventricular energy production are known to vary significantly between animals [11] but not within animals, data are presented for individual sheep. The relationship between $LVVo_2$ and P-VA for each sheep before and after ischemia is presented in Table 3; the relationship is illustrated for 1 sheep in Figure 2. Although slopes differed before and after ischemia and between sheep, correlation coefficients were good (as were the coefficients of determination) and indicated a strong association be-

Table 3. Regression of Left Ventricular Oxygen Consumption on Systolic Pressure–Volume Area

	Preischemia				Postischemia			
Sheep No.	No. of Measurements	Slope (μL O_2/ mm Hg × mL)	y-Intercept (μL O_2/ 100 g × beat)	r	No. of Measurements	Slope (μL O_2/ mm Hg × mL)	y-Intercept (μL O_2/ 100 g × beat)	r
1	5	0.056	54.2	0.91	7	0.211^a	23.2	0.93
2	7	0.227	49.2	0.95	5	0.213	59.4	0.94
3	13	0.039	41.3	0.73	8	0.051^a	89.8	0.61
4	5	0.052	28.5	0.68	9	0.243^a	29.2	0.89
5	7	0.016	11.7	0.98	8	0.071^a	8.4	0.86
6	11	0.029	28.6	0.66	6	0.044^a	15.5	0.79
7	7	0.06	41.4	0.82	11	0.16^a	38.6	0.85

[a] Significance: $p < 0.05$ compared with preischemic value.

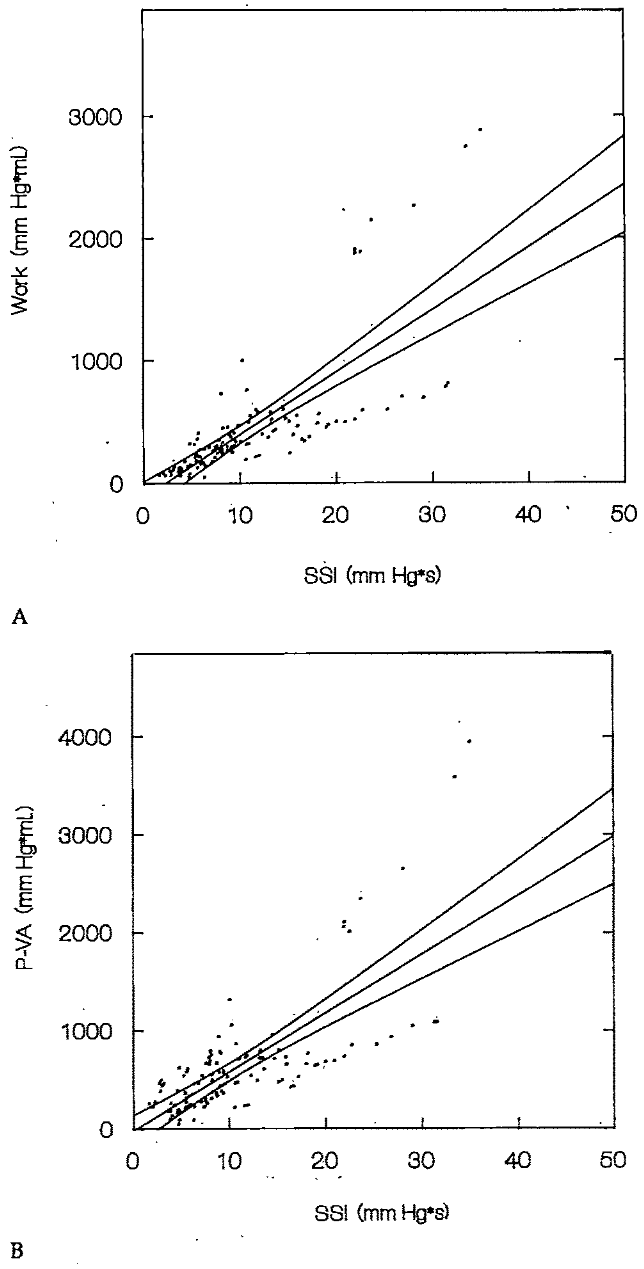

Fig 1. (A) Regression of stroke work on systolic stress integral (SSI): Y = 51.0X − 112.7; n = 123; r = 0.76. (B) Regression of systolic pressure–volume area (P-VA) on SSI: Y = 60.1X − 17.7; n = 123; r = 0.74.

tween $LVVo_2$ and P-VA. In 6 of the 7 sheep, ischemia significantly increased the slope of the relationship. The exception (sheep 2) had severely depressed left ventricular function during preischemic measurements. Intercepts varied widely, but ischemia did not cause significant differences.

Calculation of systolic pressure–volume area requires determination of V_0 by extrapolation of the Ees line. Mean V_0 before ischemia was 11.0 (standard error, ± 6.2) (n = 65) and after ischemia, 15.3 (± 6.7) (n = 58) ($p < 0.05$).

Tables 4 and 5 present the relationship between $LVVo_2$ and SSI [7, 10] and stroke work, respectively. Correlation coefficients for individual regression equations were good for both indices of left ventricular energy production, but, in general, were not as good as for the $LVVo_2$/P-VA relationship. The coefficients of determination for the $LVVo_2$/P-VA relationship were somewhat higher than those for SSI and stroke work.

Both P-VA and $LVVo_2$ can be converted to joules to determine oxygen utilization efficiency before and after ischemia (Table 6). As documented in Table 6, reversible global ischemia profoundly reduced the oxygen utilization efficiency for both external work and total mechanical energy of the ventricle (P-VA).

Comment

The present study establishes the feasibility of using systolic P-VA to measure ventricular energy production both in vivo and in postischemic, poorly contracting hearts. The coefficients of determination for regression of $LVVo_2$ on P-VA are similar for preischemic and postischemic hearts, and indicate that P-VA "explains" most of the variation in $LVVo_2$ during both experimental conditions within individual sheep.

Several methodological developments were necessary before nearly simultaneous measurements of P-VA and $LVVo_2$ could be made in vivo. The unique anatomy of the ovine coronary arterial circulation [8] permits calculation of $LVVo_2$ rather than myocardial oxygen consumption. The ovine left coronary artery exclusively supplies the left ventricle, and there is minimal overlap with the right ventricle [8]. A transit-time ultrasonic flow probe on the left main coronary artery and a fiberoptic oximeter in the coronary sinus produce nearly continuous measurements of $LVVo_2$. Three-axis sonomicrometry measurements of left ventricular dimensions provide a means to observe ventricular volume changes throughout the cardiac cycle

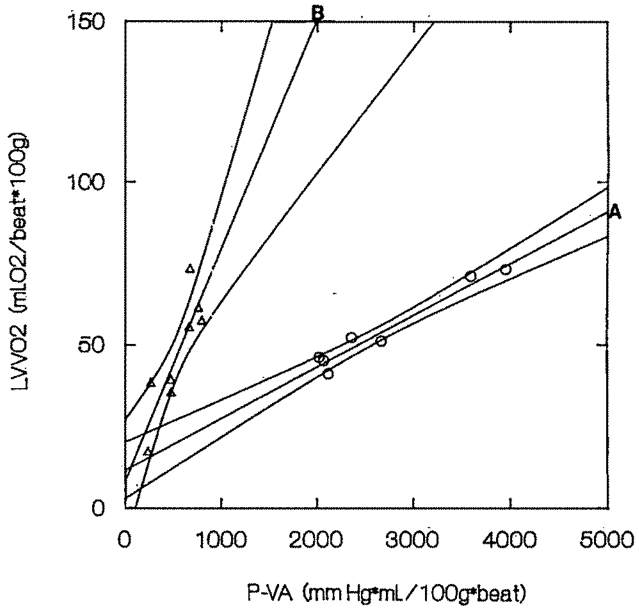

Fig 2. Regression of left ventricular oxygen consumption ($LVVo_2$) on systolic pressure–volume area (P-VA) for sheep 5 (see Table 3).

Table 4. Regression of Left Ventricular Oxygen Consumption on Systolic Stress Integral

	Preischemia				Postischemia			
Sheep No.	No. of Measurements	Slope ($\mu L\ O_2/$ mm Hg × mL)	y-Intercept ($\mu L\ O_2/$ 100 g × beat)	*r*	No. of Measurements	Slope ($\mu L\ O_2/$ mm Hg × mL)	y-Intercept ($\mu L\ O_2/$ 100 g × beat)	*r*
1	5	3.1	104.3	0.49	7	7.5[a]	33.9	0.51
2	7	6.2	−5.3	0.87	5	8.2	−11.2	0.95
3	13	1.1	43.2	0.86	6	11.9[a]	40.8	0.63
4	5	1.7	37.4	0.52	9	16.6[a]	20.8	0.07
5	7	2.2	−4.3	0.96	8	4.9[a]	21.3	0.72
6	11	1.9	22.3	0.83	6	20.4[a]	−9.8	0.82
7	7	1.2	41.2	0.30	11	2.1	33.6	0.78

[a] Significance: $p < 0.05$ compared with preischemic value.

and to generate pressure–volume loops. Last, the development of a rapid, simple method to measure single-beat pressure–volume loops at different end-systolic volumes [6] allows rapid calculations of Ees, which is a prerequisite for calculation of P-VA. Thus, technical advances from many sources are brought together for these in vivo measurements of $LVVo_2$ and P-VA in "stunned" hearts.

Compared with stroke work and SSI, P-VA has advantages as an index of left ventricular energy production. Both external work and the elastic potential energy stored in the ventricle at end-systole are separately measured and can be compared. The units of measurement are easily converted to energy units that are easily related to oxygen consumption to calculate oxygen utilization efficiency. Last, the correlation coefficients between $LVVo_2$ and the other two indices of ventricular energy production are, in general, slightly higher for P-VA than for either SSI or stroke work.

Our measurements of oxygen utilization efficiency for external work after 25 minutes of global ischemia indicate a nearly fourfold decrease from preischemic measurements. The magnitude of this decrease is similar to the fourfold increase in the regression slope of $LVVo_2$ on SSI for pooled data from 20 sheep reported previously [5]. However, the decrease in oxygen utilization efficiency for total mechanical energy production (P-VA) is only twofold after ischemia. Partition of postischemic P-VAs shows a decrease in external work and an increase in elastic potential energy in 6 of the 7 sheep; this shift in the partition of total ventricular mechanical energy explains the greater loss of oxygen utilization efficiency for external work than for total mechanical energy production.

Preischemic measurements of oxygen utilization efficiency for both external work and total mechanical energy production are within the low range of those measured by Sagawa and colleagues [3] in ex vivo cross-circulated canine hearts and in those reviewed by Gibbs [1]. In the present studies, surgical manipulation of the heart before ischemia reduced myocardial performance. Low volumes for oxygen utilization efficiency and reduced contractility suggest that surgical manipulation can also stun myocardium. This suggestion is unproved, however, as data from normal control hearts that have not been manipulated are not available.

The biochemistry of reversible myocardial ischemia is only partially understood. Current work implicates production of oxygen free radicals [12] and abnormalities of calcium function [13] within stunned myocardium. Preliminary work indicates that reperfusion with calcium blockers [14] or low-calcium perfusates [13] or oxygen free-radical scavengers [15] may reduce the contractile dysfunction associated with stunned myocardium. The

Table 5. Regression of Left Ventricular Oxygen Consumption on Stroke Work

	Preischemia				Postischemia			
Sheep No.	No. of Measurements	Slope ($\mu L\ O_2/$ mm Hg × mL)	y-Intercept ($\mu L\ O_2/$ 100 g × beat)	*r*	No. of Measurements	Slope ($\mu L\ O_2/$ mm Hg × mL)	y-Intercept ($\mu L\ O_2/$ 100 g × beat)	*r*
1	5	0.04	72.2	0.85	7	0.28	−29.1	0.89
2	7	0.3	−12.1	0.96	5	0.24	−22.3	0.92
3	13	0.13	42.8	0.65	6	0.29	44.2	0.96
4	5	0.068	30.1	0.72	9	0.243[a]	42.1	0.94
5	7	0.03	−14	0.98	8	0.161[a]	16.2	0.91
6	11	0.034	31.5	0.49	6	0.401[a]	32.4	0.78
7	7	−0.1	42.2	0.14	11	0.047[a]	38.1	0.74

[a] Significance: $p < 0.05$ compared with preischemic value

Table 6. Oxygen Utilization Efficiency

	Preischemia			Postischemia		
Sheep No.	No. of Measurements	EW/LVVo$_2$ (%)	P-VA/LVVo$_2$ (%)	No. of Measurements	EW/LVVo$_2$ (%)	P-VA/LVVo$_2$ (%)
1	5	5.5	5.9	7	3.2	4.1
2	7	2.5	3	5	3.7	8.1
3	13	3.8	5.4	6	0.4	1.5
4	5	4.9	8.8	9	1.3	4.5
5	7	25.3	27.6	8	1.9	6.7
6	11	4.3	5.6	6	1.1	1.2
7	7	2.3	3.3	11	1.3	2.5
Mean	7.9	7.0	7.6	6.9	1.8	3.8
SE	3.0	3.1	2.9	2.4	0.7	1.4

EW = external work; LVVo$_2$ = left ventricular oxygen consumption; P-VA = systolic pressure–volume area; SE = standard error.

observed effects of ischemia on oxygen utilization efficiency reported here are consistent with both production of free oxygen radicals and a defect in calcium-mediated excitation-contraction coupling [16].

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DISCUSSION

DR IRVIN KRUKENKAMP (Boston, MA): Some of you may know that Drs Levitsky, Silverman, and I have been involved in assessing myocardial oxygen consumption as a sensitive indicator of postischemic injury after cardioplegic arrest as well as normothermic stunning as has been presented here. Indeed, in a paper in the *Journal of Thoracic and Cardiovascular Surgery*, March 1988, we investigated a series of canine hearts subjected to an injury similar to that presented here today; specifically, 20 minutes of normothermic ischemia by aortic cross-clamping while on total bypass, followed by 30 minutes of reperfusion.

We assessed myocardial oxygen consumption in that group against P-VA as well as an alternative index of contractility, namely, Rankin's preload recruitable stroke work function, and we found a 20% increase in the oxygen utilization for basal metabolism. We postulate that that was necessary for reparative processes in the stunned myocardium.

In contrast to the present study, however, we found no change in the slope data for those correlations. Moreover, we investigated a similar relationship in the cardioplegic myocardium. Two hours of crystalloid or oxygenated crystalloid cardioplegic ischemia produced a similar increment in the oxygen utilization for basal metabolism.

Interestingly, blood cardioplegia prevented that increased oxygen consumption, as did augmenting the crystalloid cardioplegia with either histidine buffer or with superoxide dismutase and catalase.

I reiterate this information to show that we think that a change in y-axis intercept is a sensitive marker for postischemic injury in that we can ameliorate that change by different modalities.

Dr Furukawa, what do you think of this information in light of your studies, and have you proceeded with subsequent studies to alter this change in slope that you have observed?

DR FURUKAWA: One of the problems is that to get the y-intercept on that $LVVo_2$ versus P-VA diagram requires a large amount of extrapolation because most of the data that are obtained in vivo, not in cross-circulated dog models, are shifted to the right and you cannot measure a P-VA of zero, at least in an in vivo experiment. So most of that y-intercept is from extrapolation.

And yes, in the diagram I showed, the y-intercept was not significantly different, but that represents a large amount of error in the measurements. Therefore, I pointed that out, but I cannot make any conclusions about the y-intercept because of the way it is measured or extrapolated.

DR KRUKENKAMP: We have now recently moved to Boston from Chicago and have become interested in energy transduction, and you have shown efficiency data for consumed oxygen to P-VA. There is likewise an efficiency of converting the energy stored in the mechanical P-VA to either external work or internal work.

In a series of animals subjected to the same ischemic insult or an alternative group of dogs subjected to calcium chloride infusion, we investigated this energy transduction from P-VA to external mechanical work. With ischemia there is a decrease in this relationship as opposed to with a positive inotropic agent, which increases the ratio.

Change is accompanied by a reciprocal change in the internal work to P-VA efficiency; that is, with ischemia there is an increase in the ratio and with the positive inotrope there is a decrease.

I'm sure you have all this data on your hard disk, but I am curious if you have calculated similar information and if you have not, please do and send it to me in Boston. I am very interested to know.

DR J. SCOTT RANKIN (Durham, NC): This is a very nice study, and we have seen pretty much the same thing, but cannot figure it out exactly. If you look at your oxygen consumption versus P-VA relationship, the oxygen consumption range stays about the same, and P-VA goes way down after ischemia. That accounts for the change in slope. Now, we are pretty happy with the validity of P-VA as an index of oxygen consumption in the normal heart, but is it in fact a valid model to be using during ischemia, and how could we possibly test that hypothesis?

DR FURUKAWA: We have not tested the relationship of P-VA to $LVVo_2$ in this model with inotropic agents or any other medical or drug manipulations. So, to answer your question, I am not certain that this will hold up if further experimentation is done.

DR EDMUNDS: Let me make a comment. That is why we did the coefficients of determination, just to see how much of the $LVVo_2$ was explained by P-VA. Though not shown, we also did this for SSI and stroke work, and the highest correlation was with P-VA in the stunned or postischemic myocardium.

Dr Furukawa did show a slide on oxygen utilization for external work, and it showed that only 2% of the oxygen consumed is actually turned into external work after ischemia.

DR DIMITRI NOVITZKY (Oklahoma City, OK): I understand that the definition of stunned myocardium was introduced and coined by Eugene Braunwald. In this experiment, you are presenting a combination of two interventions: cardiopulmonary bypass and global myocardial ischemia. During cardiopulmonary bypass, a major neuroendocrine response occurs, such as the release of endogenous and systemic catecholamines, that, by itself, will induce metabolic changes. The myocardial ischemia is the second insult. The combination of these two interventions makes it very difficult to analyze the data and draw conclusions.

The other point I would like to make is that you have observed less work for the same amount of oxygen utilization. This may be a reflection of myocyte injury, such as mitochondrial, or of the presence of futile metabolic cycles. Have you measured myocardial lactate extraction?

DR FURUKAWA: Well, to answer your first question, the problem we had, as I said, was that we experimented on 22 sheep and obtained complete data in 7. Twenty-five minutes of cross-clamping produced such a severe injury that, even with manipulation of drugs, we were not always able to obtain postischemic data. So we have also gone on to do experiments with a lesser cross-clamp time.

We have also realized that people have questions regarding the effects of cardiopulmonary bypass alone without the cross-clamping or stunning effect, and we have gone on to do experiments in which we have control models that undergo cardiopulmonary bypass without stunning. The preliminary data show that there appears to be no difference or a minimal effect of cardiopulmonary bypass, and that we can separate the difference between stunning and cardiopulmonary bypass.

DR NOVITZKY: Have you observed any abnormalities regarding lactate utilization by the heart?

DR. FURUKAWA: We have not measured lactate levels. One of the problems we had was to try to obtain all of these data on an on-line basis and when you measure lactate levels, it is hard to determine what that level that you have obtained at point B represents. It is not an instantaneous measurement, but rather reflects a condition of so many minutes earlier.

DR NOVITZKY: Finally, have you performed histopathological examination of the hearts at the end of the experiment to rule out the presence of myocardial necrosis, mainly in the subendocardial area?

DR FURUKAWA: Unfortunately, we have not.

DR CORRADO MARINI (Brooklyn, NY): Because you used a model that develops pulmonary hypertension, which in turn causes right ventricular dilatation with leftward septal shift leading to decreased left ventricular compliance, I wonder if the change in the left ventricular pressure–volume slope could have been determined by a decreased septal to free wall axis.

DR FURUKAWA: We have tried to control for right heart injury by placing an ice pack on the right ventricle during the ischemic period. Granted, that is not proof, but that is the best we could do at that time, and I do not have an answer as to whether that has an effect on that slope relationship.

Reoperation for Thymoma: Report of 23 Cases

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Twenty-three patients underwent reoperation for thymoma at intervals of 2 months to 17 years 10 months after the initial operation. There were no operative or hospital deaths. Myasthenia gravis occurred in 12 patients, but in only 2 was it a determinant for reoperation. The longest survival after reoperation is 12 years 9 months, and that patient is free from tumor. Four distinct surgical groups emerged, and their recognition provides an improved method of reporting and suggests a strategy for better overall management. Group 1 (n = 5) had completion of thymectomy (reoperation) after thymomectomy alone or after incomplete thymectomy. The interval was 2 months to 17 years 10 months. All 5 had myasthenia gravis. At reoperation, thymomas were found in 3 and a hyperplastic thymus in 2. Four are alive and tumor free 2 years to 8 years 2 months after reoperation. One died tumor free after 5 years. Group 2 (n = 8) had reoperation for recurrent thymoma after standard (presumably complete) resection. The interval was 2 years to 13½ years. Four had myasthenia gravis. Four are alive 8 months to 5 years 8 months after reoperation, 3 without detectable tumor. Four died 3 years 3 months to 8 years 4 months after reoperation, 3 free from tumor. Group 3 (n = 8) underwent reoperation for initially unresectable thymoma after adjuvant treatment with chemotherapy, radiotherapy, or both. The interval was 3 months to 4 years 8 months. Three had myasthenia gravis. Six are alive 4 months to 4 years after reoperation, only 1 with tumor. Two died 1 year 5 months and 6½ years after reoperation, both with tumor. Group 4 (n = 2) underwent reoperation for miscellaneous reasons. The interval was 2 months and 3½ years. Neither had myasthenia gravis. One had thymoma at reoperation and is alive, tumor free, 12 years 9 months postoperatively. The other, reoperated on for a mediastinal mass at 3½ years, had metastatic breast cancer, not thymoma. Reoperation for thymoma can be followed by prolonged tumor-free survival. Thymomectomy alone or incomplete thymectomy should be abandoned. Recurrent thymoma is most often limited to the thorax, thus facilitating reoperation. The response of initially inoperable thymoma to adjuvant therapy favors subsequent reoperation. Slow growth and late appearance of recurrence necessitate lifelong follow-up. Myasthenia gravis, when present, had no deleterious effect on operation.

(Ann Thorac Surg 1990;49:550–5)

The role of reoperation for thymoma in the overall management of this tumor has not been clearly defined. Reoperation has been mentioned in the literature in an anecdotal manner, usually in the form of isolated case reports [1–5]. In four reports of large series [6–9] totaling 754 operations, only 27 patients (3.6%) underwent reoperation, and in many series of equivalent size, no reoperations are even mentioned. In this report, 23 patients having reoperation are classified into four distinct groups. A pattern emerges that provides an improved method of reporting and suggests a strategy for better management based on specific determinants for reoperation.

Material and Methods

In the 34-year period 1955 through July 1989, I operated on 108 patients with thymoma. A total of 114 operations were done, 23 (20%) of which were reoperations. All reoperations were performed in the 18 years since 1971. I performed the first operation in 2 patients, the second in 14, both in 6, and neither in 1 patient.* There were no operative or hospital deaths, and follow-up was 100%.

Of the 23 patients having reoperation, there were 11 male and 12 female patients with mean ages of 41.1 and 47.2 years, respectively. All were staged pathologically at the initial operation according to the classification of Masaoka and colleagues [10]. There were 7 patients in stage I, 6 in stage II, 8 in stage III, and 1 in stage IV; 1 patient had no thymoma at the initial operation.

Twelve patients had myasthenia gravis (MG) at some time in their clinical course. The complex thymus-thymoma-MG relationship played a role in only 2 patients in whom completion of thymectomy was carried out when MG appeared after enucleation of thymoma. Otherwise MG did not influence or complicate the reoperation and is not a major thrust of this report.

The 23 patients having reoperation could be classified into four distinct surgical groups.

Group 1 (n = 5): reoperation for completion of thymectomy after thymomectomy alone or after incomplete thymectomy.

Group 2 (n = 8): reoperation for recurrent thymoma after standard (presumably complete) thymectomy.

Presented at the Twenty-fifth Anniversary Meeting of The Society of Thoracic Surgeons, Baltimore, MD, Sep 11–13, 1989.

Address reprint requests to Dr Kirschner, 2 E 92nd St, New York, NY 10128.

*This patient was kindly provided by the late Dr Angelos Papatestas.

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0003-4975/90/$3.50

Table 1. Summary of Data for Group 1 (Completion of Thymectomy)

Patient No.	Sex	Age (yr)	Stage	Interval (mo)	Survival[a] (mo)	Survival[b] (mo)	Myasthenia Gravis	Current Status of Tumor	Alive
1	F	38	I	2	24	26	Yes	Negative	Yes
2	M	62	I	4	79	83	Yes	Negative	Yes
3	M	43	II	7	96	103	Yes	Negative	Yes
4	F	50	I	89	60	149	Yes	Negative	No
5	F	46	I	214	98	312	Yes	Negative	Yes

[a] This is survival after reoperation. [b] This is total length of survival.

Group 3 (n = 8): reoperation for initially unresectable thymoma after adjuvant treatment with chemotherapy or radiotherapy or both.

Group 4 (n = 2): reoperation for miscellaneous reasons.

In this study, reoperation means any major procedure for recurrent or initially unresectable thymoma, with or without MG, after prior surgical intervention, be it biopsy, exploratory thoracotomy, or partial or complete resection. Patients having simple biopsy merely for tissue diagnosis followed immediately by major intervention are not considered as undergoing reoperation. However, if biopsy led to adjuvant therapy before definitive major surgical intervention it is included. One patient with initially nonthymomatous MG was included because thymoma was found at reoperation.

Results

Group 1: Reoperation for Completion of Thymectomy

Four of the 5 patients initially had enucleation of thymoma, and the fifth had a transcervical thymectomy (the first patient in the Mount Sinai series [11], deemed here to have had an incomplete thymectomy). Four were in stage I and 1, stage II (Table 1). Reoperation was carried out early in 3 patients at 2, 4, and 7 months and late in 2 at 7 years 5 months and 17 years 10 months. One patient had MG before the first operation; in the other 4, it developed only after the initial operation. All 5 had MG at reoperation.

Incisions for the first operation were cervical in 2 patients (one for a cervical thymoma and the other for a transcervical thymectomy) and a posterolateral thoracotomy in the other 3 (all for enucleation of thymoma). All 5 underwent reoperation through a median sternotomy, with removal of hyperplastic nontumorous thymus in 2 and multiple thymomas in residual thymus in 3.

Survival after reoperation ranged from 2 years to 8 years 2 months. Four patients are alive and free from tumor. One has severe MG, 2 have very mild MG, and 1 is in complete remission. The patient who died at 5 years still had MG but was tumor free. No patient received either chemotherapy or radiotherapy. The patient who has survived the longest after the initial operation is free from tumor and is in complete remission from MG 26 years later.

Group 2: Reoperation for Recurrence After Complete Thymectomy

Seven patients had thymoma at the initial operation. Three were in stage I, 1 was in stage II, 2 were in stage III, and 1 was in stage IV (Table 2). One patient had nonthymomatous MG. Six patients were operated on initially through a median sternotomy; 1, through a posterolateral thoracotomy; and 1, through a cervical incision.

Complete resection was believed to have been carried out in 5 patients. Tiny fragments of unresectable tumor were left behind in 2. No tumor was found in the transcervical thymectomy specimen at the first operation. Radiotherapy was given postoperatively to only 1 patient.

Table 2. Summary of Data for Group 2 (Reoperation for Recurrence)

Patient No.	Sex	Age (yr)	Stage	Interval (mo)	Survival[a] (mo)	Survival[b] (mo)	Myasthenia Gravis	Current Status of Tumor	Alive
1	F	51	I	24	28	52	No	Negative	Yes
2	M	28	IV	29	100	129	No	Negative	No
3	M	47	0	29	39	68	Yes	Negative	No
4	F	57	II	38	68	106	No	Positive	Yes
5	M	32	III	45	22	67	Yes	Negative	Yes
6	M	55	III	132	8	140	Yes	Negative	Yes
7	M	55	I	132	60	192	Yes	Positive	No
8	F	50	I	162	69	231	No	Negative	No

[a] This is survival after reoperation. [b] This is total length of survival.

Table 3. Summary of Data for Group 3 (Reoperation After Adjuvant Therapy)

Patient No.	Sex	Age (yr)	Stage	Interval (mo)	Survival[a] (mo)	Survival[b] (mo)	Myasthenia Gravis	Current Status of Tumor	Alive
1	M	24	II	3	48	51	No	Negative	Yes
2	F	59	II	3	43	46	Yes	Positive	Yes
3	M	51	III	6	21	27	Yes	Negative	Yes
4	F	26	III	7	4	11	No	Negative	Yes
5	F	49	III	11	28	39	No	Negative	Yes
6	M	28	III	12	17	29	No	Positive	No
7	F	45	III	13	10	23	Subclinical	Negative	Yes
8	M	27	II	56	78	134	Yes	Positive	No

[a] This is survival after reoperation. [b] This is total length of survival.

Myasthenia gravis was present initially in 3 patients, and it developed for the first time after reoperation in 1.

At reoperation (2 to 13½ years later), 7 patients had a lateral thoracotomy, and 1 had a repeat cervical incision for an intrathyroid cervical thymoma stage II. Recurrent disease was resected from the mediastinum in 3, pleura in 3, lung in 1, and neck in 1.

Four patients are alive, 3 without detectable tumor, 8 months to 5 years 8 months after reoperation. Two have mild MG. Four patients died 3 years 3 months to 8 years 4 months after reoperation (overall survival after initial procedure, 5 years 8 months to 19 years 3 months), only 1 with residual tumor and 2 with MG. The patient with cervical thymoma died in MG crisis 3 years 3 months after reoperation; no tumor or residual thymus tissue was found at autopsy.

Group 3: Reoperation After Adjuvant Therapy

More-advanced stages predominated in this group. No patient was in stage I, 3 were in stage II, and 5 were in stage III (Table 3). Three had MG. The initial operation was performed through a lateral thoracotomy in 2, a median sternotomy in 2, an anterior parasternal mediastinotomy in 2, a cervical mediastinoscopy in 1, and needle biopsy of a pleural mass in 1.

Chemotherapy alone was given in 4 patients, radiotherapy alone in 2, and both in 2. Five patients underwent planned reoperation after adjuvant therapy at intervals of 3 to 13 months, and the remaining 3 had reoperation without a formally planned adjuvant program at 3 months, 1 year, and 4 years 8 months after the initial operation.

Six patients are alive 4 months to 4 years after reoperation; only 1 has recurrent thymoma as well as MG. Another patient is in complete remission from MG. Two patients, both with persistent thymoma, died 1 year 5 months and 6½ years after reoperation.

Group 4: Miscellaneous Reoperation

One patient had reoperation 2 months after the initial limited anterolateral thoracotomy with inadequate exposure. At reoperation through a generous right posterolateral incision, en bloc resection including right pneumonectomy for a stage III thymoma was accomplished. A tiny shred of tumor was left on the junction between the left innominate vein and the superior vena cava. Postoperative radiotherapy and chemotherapy were given, and the patient is alive, symptom free, and tumor free 12 years 9 months after reoperation.

The other patient initially underwent simultaneous right modified radical mastectomy for coexisting breast cancer and median sternotomy through the same incision (under the mastectomy flaps) for stage II thymoma. Three and one-half years later at reoperation a mediastinal mass was noted, which proved to be metastatic breast cancer, not recurrent thymoma.

Neither patient had MG.

Comment

Reoperation for thymoma appears to be a function of one or more of the following "determinants."

Anatomy and Embryology of Thymus: Ectopic Thymus Tissue

Two of the 23 patients had cervical thymomas, illustrating ectopic location of the thymus in the neck (failure of descent). Both patients were seen with neck masses clinically deemed to be of thyroid origin. Myasthenia gravis developed in 1 (group 1) after the cervical thymoma was removed. Thymectomy was completed later through a sternum-splitting incision. Only hyperplastic nontumorous thymus was removed, with some improvement of the MG.

The other patient (group 2) initially had a transcervical thymectomy for MG. Only hyperplastic thymus was removed, and the thymectomy was thought to be complete. Twenty-nine months later a cervical mass appeared, which proved at reoperation to be a stage II thymoma in the left thyroid lobe. Thus, even though a cervical incision was used for the original thymectomy, ectopic undescended thymus tissue (tumor ?) was initially overlooked in the neck and subsequently gave rise to the cervical mass. At autopsy 3 years 3 months later (death from MG crisis), no residual thymus tissue or thymoma was found in the neck or mediastinum.

Pathology of Thymoma: Microscopic, Gross, and Surgical Stage

It is generally believed that gross appearance and behavior of thymoma at operation are indicative of benign or malignant potential. This is the basis for surgical staging. However, recurrence of noninvasive stage I thymoma has been reported [2]. In the current series, there were four stage I tumors in group 1 patients and three in group 2. Two of the former, originally enucleated, recurred with multiple tumors in residual thymus at reoperation (completion of thymectomy), whereas all three in group 2 recurred despite initial presumably complete thymectomy through an adequate median sternotomy.

There are two possible explanations for such recurrences. The first is the multicentric nature of some thymomas. Bernatz and associates [12] reported three such cases in a series of 123 thymomas, and Jaretzki and co-workers [13] mentioned finding two distinct histologically different thymomas in separate mediastinal lobes. Castleman and Norris [14] emphasized the tendency for thymoma to exist in lobules. Thus, unresected residual thymus tissue can be a potential site for the development of additional thymomas at a later date, or it can harbor multiple small tumors or lobules of tumors not detected at the initial operation, especially if surgical exposure is inadequate or if simple enucleation is done.

The second explanation may lie in recent reevaluation of microscopic findings. Nomori and colleagues [15] performed morphometric studies of the nuclei of the tumor and showed a correlation between increased nuclear diameter and malignant potential. Marino and Müller-Hermelink [16] and Ricci and co-authors [17] have shown that thymomas can arise from epithelial cells of either the cortex or the medulla of the thymus. Despite identical surgical staging (stage I), the former have malignant potential, and the latter do not recur.

Pattern of Recurrence: Local, Regional, Distant

The propensity for local and intrathoracic recurrence and the rarity of distant hematogenous and lymphatic spread make reoperation for such recurrence a valid therapeutic measure. In 7 of the 8 patients in group 2, recurrent disease was resected with tumor-free survival ranging from 8 months to 8 years 4 months in 5 patients.

Slow Growth: Long Life History

Thymomas usually grow very slowly, and recurrences may not appear for many years after the initial operation. Thus follow-up should continue indefinitely. For the 23 patients in this series, the longest interval between initial procedure and reoperation was 17 years 10 months, and that patient remains alive, well, and tumor free 8 years 2 months after reoperation.

Response to Adjuvant Therapy: Chemotherapy, Radiotherapy, or Both

Experience with combination chemotherapy consisting of bleomycin sulfate, Adriamycin (doxorubicin hydrochloride), cisplatin, and prednisone (BAPP) has been shown to be effective in patients with locally advanced unresectable thymoma [18]. The patients in group 3 represent such a group. A prospective planned program of BAPP therapy followed by reoperation resulted in regression of initially widespread unresectable tumor to a relatively localized state, thus enabling a reasonably complete excision of residual tumor at reoperation. Such chemotherapy does not preclude radiotherapy, the traditional form of adjuvant treatment. The longest survival times after reoperation in this group are 4 years and 3 years 7 months. It is expected that this group of patients will grow in numbers and importance in the future.

Surgical Technique: Adequacy of Exposure and Completeness of Thymectomy

Some reoperations were done because of inadequate surgical technique at the first operation. These include completion of thymectomy (all 5 patients in group 1) and consummation of extensive extended resection through wide surgical exposure in 1 patient in group 4 whose disease was declared inoperable elsewhere because of limited exposure initially. This patient underwent combined en bloc thymectomy and right total pneumonectomy for a stage III thymoma. Postoperative radiotherapy and chemotherapy were given. The patient is alive and tumor free 12 years 9 months after reoperation.

In my opinion, a wide exposure—complete sternal splitting with opening of both pleural cavities—is indicated in all patients with thymoma however limited in extent so as to accomplish as complete a thymectomy as possible. If this principle is adhered to, group 1 would cease to exist in a classification of reoperation.

Myasthenia Gravis

The problem of the thymus-thymoma-MG relationship is complex and will be discussed only briefly here in the context of this experience with reoperation. In group 1, MG appeared for the first time after the initial operation in 4 patients from the immediate postoperative period to more than 17 years later. At reoperation, 2 patients had recurrent thymomas, and 2 had only hyperplastic residual thymus when thymectomy was completed. In 1 patient in group 2, MG developed only after reoperation for recurrence.

It has been glibly suggested that postoperative MG is due to failure to remove all nonneoplastic thymic tissue. However, in an extensive review, Namba and co-workers [19] showed that most such patients were not found to have residual thymus at postmortem examination or reoperation. In the patient of Madonick and colleagues [20], completion of thymectomy had no effect on MG. On the other hand, Strieder [21] reported complete remission in such a patient after completion of thymectomy.

This peculiar behavior may be related to the unique nature of MG in patients with thymoma. Recent studies in immunobiology by Compston and associates [22] suggest that MG with thymoma is different from nonthymomatous MG based on titers of antibodies, histocompatibility antigens, and other immunological variables. They suggest a new classification of MG as follows: (1) MG with thymoma; (2) MG without thymoma, age of onset less than 40 years; and (3) MG without thymoma, age of onset greater than 40 years. All three groups have distinct immunological spectra and sex distribution. Compston and associates intimate that MG with thymoma may be a less "thymus-dependent" form of the disease than the other two types.

In the series of Jaretzki and co-workers [13] involving 15 patients with thymoma and MG who underwent "maximal" thymectomy, 2 patients died late postoperatively of myasthenic crisis. This phenomenon did not occur in any of the patients with nonthymomatous MG having the same operation.

Despite these curious phenomena, it is recommended that the surgeon perform as complete a thymectomy as possible regardless of how limited or localized the thymoma might be, even if there is no MG at the time of operation.

Myasthenia gravis occurred in only 12 of the 23 patients in this series. In no instance did it complicate the reoperation. Because the thrust of this report is reoperation for thymoma, MG was not further analyzed nor is the Osserman classification [23] applicable.

Conclusions

Seven conclusions can be drawn from this series.

1. Reoperation is a valid accessory to an unsuccessful initial operation.
2. Reoperation is based on anatomical, pathological, histological, and behavioral features of thymus and thymoma.
3. Thymomectomy alone (enucleation) or incomplete thymectomy should be abandoned in favor of "complete" thymectomy. Wide operative exposure is essential.
4. Response of initially inoperable thymoma to chemotherapy or radiotherapy or both favors successful reoperation. I prefer chemotherapy.
5. The presence of MG does not have a deleterious effect on reoperation.
6. Slow growth and late recurrence of thymoma necessitate lifelong follow-up of all patients.
7. Reoperation is safe.

I am grateful to Richard DeAsla for help in preparing graphic material and to Sally Lucas for secretarial assistance.

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DISCUSSION

DR EARLE W. WILKINS, JR (Williamstown, MA): It was indeed a pleasure to review this unique, impressive, and very interesting manuscript. It is unique because it is by a single author; it is impressive because in his experience with 23 reoperations, the author believes—and I concur after a brief review of the literature—that this is the largest recorded series. And it is especially interesting to me because the author's experience encompasses much of the historical experience with operation for thymoma.

It is important, I believe, to translate experiential lessons, which are so well illustrated in this paper, into guidelines for current application. The ultimate aim, of course, should be to minimize the need for such reoperations.

Would Dr Kirschner accept four guidelines, which I perhaps have oversimplified from his manuscript? First, the surgical approach for thymoma should be through a total mediastinotomy, never just a transcervical approach, and then opening both pleural membranes for accurate assessment of the extent of disease and appropriate exposure for total excision.

Second, the thymectomy should always be total. Tumor is not infrequently found microscopically in apparently normal adjacent thymus, even in those tumors that appear grossly encapsulated.

Third, in patients in pathological stages II and III, postoperative adjuvant therapy is indicated, most often I believe with radiation therapy in a dose of about 5,000 rads.

Fourth, for grossly large and extensive, possibly unresectable, tumors, which are now so nicely defined by computed tomography and magnetic resonance imaging, initial biopsy should be followed by irradiation or chemotherapy or both before a definitive effort at excision.

I heartily endorse Dr Kirschner's philosophy that reoperation for recurrent thymoma is worthwhile. I do have a small quibble with his inclusion of 4 of the patients in group 3. These were the 3 patients who had a mediastinotomy and the 1 patient who had needle biopsy for diagnosis. In his words, these were "merely for tissue diagnosis." I would say they were part of a comprehensive planned treatment program, and in that sense, they do not seem to me to represent reoperations, certainly not recurrences.

Finally, using this latter interpretation, in our experience at Massachusetts General Hospital with 160 patients with thymoma, there have been just 5 patients who underwent reoperation. One was a patient in whom a small thymoma was missed at a prior cervical thymectomy elsewhere. One was a patient of mine for whom I was not able to accomplish anything at reoperation, and 3 patients benefited by total removal of recurrent disease and continuing survival. These 3 were, by the way, your patients, Dr Grillo.

DR KIRSCHNER: Dr Wilkins, thank you very much for your flattering remarks. I have studied your reports in great detail and have learned much. In regard to your questions, I hold with you completely as follows: First, a complete median sternotomy with wide opening of both pleural cavities should be done no matter how small or limited the thymoma. This facilitates maximal thymectomy as well as detection of areas of spread, which are sometimes not demonstrable by the most sophisticated preoperative studies now available.

Second, as total ("complete") a thymectomy as possible should be done as an initial procedure, not merely an enucleation of an encapsulated thymoma or an equivalent limited thymic resection. This will address the pathological phenomenon of multicentricity. Also, it will deal with the complex problem of MG, which can be present before or can develop after operation and which, in some instances, may be related to residual thymic tissue.

Third, the role of postoperative radiotherapy for malignant thymoma, completely or incompletely resected, is difficult to establish. There is no predictable response pattern. Radiotherapy has been used widely and for many years, and even seems to be effective in some instances. However, based on the salutary clinical and pathological effects of chemotherapy noted in patients undergoing reoperation, perhaps this form of treatment should begin to displace traditional radiotherapy. Subsequent radiotherapy is not precluded by this approach.

Fourth, in regard to my group 3, some reoperations (the more recent) were prospectively planned when the beneficial response to chemotherapy began to be noted. For this reason, when obvious unresectability could be determined initially by such limited procedures as mediastinoscopy or even simple needle biopsy, planned formal surgical intervention after chemotherapy was considered to be a "reoperation."

The follow-up is relatively short in group 3 because of its more recent adoption as a treatment category. Longer follow-up, more patients, and as yet undiscovered and untried chemotherapy programs will help establish the validity of this approach.

Retrograde Coronary Sinus Cardioplegia for Aortic Valve Operations: A Clinical Report on 500 Patients

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Retrograde delivery of cardioplegic solutions has recently been the subject of renewed interest, but the reliability of this technique has not been assessed in large clinical series. From 1980 to 1989, we used retrograde coronary sinus perfusion as the exclusive means of cardioplegia delivery in 500 consecutive patients undergoing aortic valve replacement, either isolated (359 patients) or combined with another valve or coronary procedure (141 patients). The coronary sinus was always cannulated under direct vision after bicaval cannulation with snaring. Cold crystalloid cardioplegia was delivered retrogradely at an average flow rate of 100 mL/min in conjunction with topical and systemic (25°C) hypothermia. The mean cross-clamp time was 83 ± 23 minutes (± the standard deviation). There were 31 hospital deaths (6.2%), 20 of which were cardiac related. Transient hemodynamic instability (defined as a need for inotropic agents for less than 24 hours postoperatively) occurred in 16 patients (3.2%), whereas a true low-output syndrome developed in 60 patients (12%). The incidence of clinically significant supraventricular arrhythmias and of permanent conduction defects was 7.4% and 1.2%, respectively. There were three nonfatal coronary venous injuries during our early experience. We conclude that coronary sinus perfusion is a safe and effective means of delivering cardioplegia in aortic valve operations. While providing a degree of myocardial protection similar to that reported with anterograde cardioplegia, the coronary sinus technique offers distinct advantages, in particular, the avoidance of perfusion-related coronary artery complications and the opportunity to repeat cardioplegia administration without interrupting the procedure. As another attractive feature of the coronary sinus route is to ensure homogeneous distribution of cardioplegia beyond coronary artery occlusions, the present study, by establishing its reliability on a large clinical scale, supports the more liberal use of this approach in select subsets of patients having coronary artery procedures.

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Over the past years, there has been an increasing interest in the use of retrograde coronary sinus perfusion for delivering cardioplegic solutions during surgically induced ischemic arrest [1–3]. However, a survey of the literature shows that the large number of experimental studies contrasts sharply with the relative paucity of clinical data.

We started to use coronary sinus cardioplegia in 1980 in patients undergoing aortic valve replacement (AVR) for aortic insufficiency, with the primary objective of avoiding the risks inherent to direct coronary ostial perfusion. The satisfactory results yielded by this technique subsequently led us to extend its use to all procedures involving the ascending aorta as well as to select subsets of coronary artery bypass operations. We report here our experience with retrograde coronary sinus perfusion as the exclusive means of cardioplegia delivery in 500 consecutive aortic valve procedures performed in our institution over a 9-year period.

Material and Methods

Patient Population

This study comprised 500 consecutive patients who underwent AVR between June 1980 and April 1989, and in whom cardioplegia was exclusively given retrogradely through the coronary sinus. Table 1 presents the main clinical characteristics of this patient population. Half of the patients were in New York Heart Association functional classes III and IV. Forty-one patients (8.2%) underwent valve replacement on an emergency basis (functional class V). Two hundred sixty-three patients (52.6%) had electrocardiographic and echocardiographic evidence of left ventricular hypertrophy [4]. In 28% of the patients, AVR was combined with another cardiac surgical procedure, in most instances, mitral valve replacement.

The low incidence of concomitant coronary artery bypass grafting is due to the fact that in the subset of patients requiring AVR and simultaneous myocardial revascularization, cardioplegic arrest was usually induced anterogradely through the aortic root and subsequently maintained by coronary sinus perfusion. These patients were excluded from this study, as it deals only with the exclusive use of the retrograde approach for delivering cardioplegic solution.

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Table 1. Summary of Patient Data[a]

Variable	Value
Mean age (yr)	53 ± 18
Age distribution (yr)	
<50	196
51–70	197
>70	107
Sex	
Male	339
Female	161
Aortic valve lesion	
Stenosis	246
Regurgitation	185
Mixed	69
Procedure	
Isolated AVR	359
AVR + other valve procedure[b]	127
AVR + CABG	14
Cross-clamp time (min)	
Isolated AVR	76 ± 18
AVR + other valve procedure	100 ± 25
AVR + CABG	114 ± 26

[a] When applicable, data are shown as the mean ± the standard deviation or as the number of patients. [b] The associated procedures were mitral valve replacement (99 patients), mitral valve replacement + tricuspid annuloplasty (27 patients), and tricuspid annuloplasty (1 patient).

AVR = aortic valve replacement; CABG = coronary artery bypass grafting.

Operative Techniques

Standard anesthetic and operative techniques were used throughout the study period. Cardiopulmonary bypass was established between the ascending aorta and the two individually cannulated venae cavae, which were snared shortly after the onset of bypass. Care was taken to insert the inferior vena cava cannula into the posterior part of the right atrial free wall, close to the interatrial groove, so that the coronary sinus orifice could be easily exposed through a short (2- to 3-cm) atriotomy made on the anterior free aspect of the right atrium, parallel to the atrioventricular sulcus.

The technique of coronary sinus cardioplegia has been previously described [5]. Briefly, the ascending aorta is cross-clamped as soon as the heart fibrillates, and a balloon-tipped catheter is placed under direct vision in the terminal portion of the coronary sinus. The balloon is gently inflated with saline solution until it occludes the coronary sinus orifice, and infusion of cardioplegic solution is initiated. The surgical procedure is then carried out in the usual way.

For several years, we used a Foley urinary catheter (12F). However, the major concern about using this device was the spherical shape of its balloon, which, once inflated, was likely to occlude the ostia of the terminal branches of the coronary sinus, thereby preventing cardioplegic solution from reaching the corresponding right ventricular myocardial areas [6].

Therefore, we designed a specific balloon (Retroperfusion Systems Inc, Costa Mesa, CA) that becomes pear shaped after inflation and thus nicely fits the funnellike configuration of the coronary sinus orifice. The upper part of the inflated balloon remains seated around the intraatrial rim of the coronary ostium, and this ensures that none of the branches of the coronary sinus is excluded from effective retroperfusion. In all of our patients, the catheter was kept secured in the coronary sinus by means of balloon inflation throughout the period of aortic cross-clamping, which allowed us to repeat cardioplegia infusions at any time without interrupting the surgical procedure. Ridges on the surface of the newly designed balloon further enhance self-maintenance of the catheter in the proper position during the arrest period.

In the early years of our experience, coronary sinus pressure was measured during cardioplegia administration through a pressure line connected to the proximal end of the Foley catheter, and was maintained in the range of 40 mm Hg. Subsequently we discontinued pressure monitoring and now simply fix the flow rate of the cardioplegia delivery system to approximately 100 mL/min, as we [7] found that under conditions of cold cardioplegic arrest, maintenance of the flow rate at that value reliably ensures that distal perfusion pressure will remain within the appropriate range.

Our protocol of cardioplegia delivery was kept constant throughout this 9-year time frame. One liter of crystalloid cardioplegic solution at 4°C was initially given over a ten- to 15-minute period. Additional doses of 500 mL of solution were reinfused in the same retrograde way every 20 minutes, yielding an average of two to three cardioplegia infusions per patient. Drainage of the cardioplegic retroperfusate was provided by the open aortic root and the right atriotomy. In no instance did the backflow of clear solution through the coronary ostia (predominantly the left coronary ostium) interfere with the surgical procedure. In addition to multidose cardioplegia, myocardial protection was provided by systemic cooling (25°C) and by topical hypothermia using continuous pericardial irrigation with cold saline solution and, more recently, ice slush. These measures allowed us to maintain myocardial temperature, usually recorded in the anterior right ven-

Table 2. Composition of Cardioplegic Solution and Reperfusion Solution

Variable	Cardioplegic Solution	Reperfusion Solution
K^+ (mEq/L)	12	15
Na^+ (mEq/L)	100	100
Mg^{2+} (mEq/L)	26	. . .
Ca^{2+} (mEq/L)	0.5	2.5
Cl^- (mEq/L)	100	100
Mannitol (g/L)	20	24.70
Glutamic acid (g/L)	2,942	2,942
pH	7.40 (at 20°C)	7.70 (at 28°C)
Osmolarity (mosm/L)	370	370

Table 3. *Hospital Mortality*

	Time Frame 1 (1980–1984) (n = 320)		Time Frame 2 (1985–1989) (n = 180)		Total (n = 500)	
Operation	No. of Patients	%	No. of Patients	%	No. of Patients	%
Isolated AVR	16/229	6.9	2/130	1.5	18/359	5.0
AVR + associated procedure	7/91	7.6	6/50	12	13/141	9.2
Total	23/320	7.1	8/180	4.4	31/500	6.2

AVR = aortic valve replacement.

tricular free wall, between 10° and 15°C during aortic occlusion.

On completion of the surgical procedure, the balloon is deflated, the catheter is removed, and the right atriotomy is closed with a continuous 5-0 polypropylene suture. One liter of a modified crystalloid reperfusate at 28°C is infused into the aortic root immediately before aortic unclamping [8]. The composition of the cardioplegic and reperfusion solutions is detailed in Table 2. Whenever necessary, defibrillation is performed shortly after restoration of coronary blood flow. The last steps of the operation then proceed in a standard fashion.

Follow-up

The postoperative records of the 500 patients were carefully reviewed for assessment of five major end points: operative mortality, requirement for pharmacological support, myocardial infarction, arrhythmias, and coronary sinus injury. Operative mortality was defined as deaths occurring during the postoperative hospital stay or within 30 days of the operation. In all instances the cause of death could be classified as cardiac related or noncardiac related. Postmortem examination of the heart was performed whenever possible and consistently included the search for left ventricular subendocardial hemorrhagic necrosis, a typical pattern of inadequate myocardial protection [9].

Analysis of the requirement for postoperative pharmacological support led to the classification of drug-dependent patients into two categories. Patients who needed moderate amounts of inotropic drugs (usually dobutamine hydrochloride, 5 μg/kg/min) for less than 24 hours postoperatively were considered as having transient postbypass hemodynamic instability; in contrast, patients who required inotropic agents or vasopressors or both for more than 24 hours postoperatively were categorized as having a true low cardiac output syndrome.

A postoperative myocardial infarction was diagnosed by the appearance of new Q waves associated with an increase in the levels of the myocardial-specific isoenzyme of creatine kinase (≥10% of the total creatine kinase content). Postoperative arrhythmias were classified into three categories: (1) sustained supraventricular (nonsinus) tachyarrhythmias of new onset necessitating antiarrhythmic therapy, with or without the need for direct-current cardioversion; (2) multifocal or coupled premature ventricular beats with a normal concentration of serum potassium necessitating antiarrhythmic drugs; and (3) ventricular tachycardia or fibrillation. Conduction defects were classified as (1) junctional rhythm, (2) bundle-branch block of new onset, and (3) complete heart block.

Statistical Analysis

For statistical analysis, the incidence of phenomena was compared by the χ^2 test. All values are shown as the mean ± the standard deviation. A p value of less than 0.05 was considered significant.

Table 4. *Cardiac-Related Mortality*

	Time Frame 1 (1980–1984) (n = 320)		Time Frame 2 (1985–1989) (n = 180)		Total (n = 500)	
Operation	No. of Patients	%	No. of Patients	%	No. of Patients	%
Isolated AVR	9/229	3.9	2/130	1.5	11/359	3.0
AVR + associated procedure	6/91	6.5	3/50	6.0	9/141	6.3
Total	15/320	4.6	5/180	2.7	20/500	4.0

AVR = aortic valve replacement.

Table 5. Postoperative Inotropic Support

		Time Frame 1 (1980–1984) (n = 320)		Time Frame 2 (1985–1989) (n = 180)		Total (n = 500)	
Indication	Duration	No.	%	No.	%	No.	%
Transient postbypass hemodynamic instability	≤24 h postop	13	4.0	3	1.6	16	3.2
Low cardiac output syndrome	>24 h postop	38	11.8	22	12.2	60	12
Total		51	15.9	25	13.8	76	15.2

Results

Operative Mortality

The data are summarized in Tables 3 and 4. Overall hospital mortality was 6.2% (31 patients). Postoperative ventricular dysfunction was the cause of death in 20 (64.5%) of these patients. Among the 20 cardiac-related deaths, six occurred in patients who had operation for acute bacterial endocarditis, which in 2 patients involved a prosthetic valve. Not unexpectedly, operative mortality was influenced by age (operative mortality rose from 2.7% for patients less than 70 years old to 18.6% for patients more than 70 years old; $p < 0.0001$), timing of operation (patients undergoing an emergent operation had a 12.1% operative mortality and those undergoing an elective procedure, 5.6%; p = not significant), and performance of associated procedures (patients who underwent isolated AVR had a 5% operative mortality, which increased to 9.2% for those who underwent a concomitant valve or coronary artery procedure; p = not significant). Further, when the data were arranged by time frame, the operative mortality was found to decrease significantly in time frame 2 only for patients having isolated AVR (1.5% versus 6.9% in time frame 1; $p < 0.03$). In contrast, the strong effect of associated procedures on hospital mortality was similar in both time frames.

Postmortem examination of the heart was performed on 18 patients, 13 of whom belonged to the group of 20 with a cardiac-related death. In only 2 patients were patterns suggestive of inadequate intraoperative myocardial protection clearly documented. In both patients, cardiopulmonary bypass had been reinstituted after the initial AVR because of bleeding problems in 1 patient and the need to relieve a previously underestimated left ventricular outflow tract obstruction in the other.

In 6 patients, death was related to a myocardial infarction caused by atherosclerotic lesions of the coronary arteries; in only 1 of these patients had bypass grafting been done. One additional patient had evidence of embolic occlusion of the left anterior descending coronary artery by atheromatous debris, which had probably migrated from a severely diseased ascending aorta. Two patients died of a technical problem (aortic dissection, 1 patient; and a fistula between the ascending aorta and the right ventricle after AVR for acute bacterial endocarditis, 1). No specific cardiac abnormality was found in the 2 remaining patients whose deaths were due to left ventricular failure. This was also true for the 5 patients who died of noncardiac causes.

Postoperative Pharmacological Support

Overall, 15.2% of the 500 patients required some form of pharmacological support postoperatively (Table 5). Sixteen patients had transient postbypass hemodynamic instability, the incidence of which was lower, although not significantly, in time frame 2 (1.6% versus 4.0% in time frame 1; p = not significant). Sixty additional patients had a true low cardiac output syndrome, an incidence of 12%, which remained fairly constant over the course of this study. In none of the patients was an intraaortic balloon or another circulatory support device inserted intraoperatively or postoperatively.

Myocardial Infarction and Arrhythmias

Seven patients sustained a perioperative myocardial infarction. This overall incidence of 1.4% decreased to 0.4% (2 patients) in time frame 2.

Thirty-seven patients (7.4%) experienced postoperative supraventricular arrhythmias, primarily atrial fibrillation (35 patients). In addition, ventricular arrhythmias developed in 12 patients (2.4%) and included clinically significant premature ventricular beats in 7 and ventricular tachycardia in 5.

Overall, 62 patients (12.4%) experienced some form of conduction defect postoperatively. Twenty-seven patients had bundle-branch block, which was complete in 15. Fourteen patients had junctional rhythm. Complete heart block was present in 21 patients; in most instances (15/21), the conduction defect was transient. Only 6 patients (1.2% of the entire series) required insertion of a permanent pacemaker.

Coronary Sinus Injury

Coronary venous injury was encountered in 3 patients (0.6%). All three injuries occurred during our early experience. In 1 patient, the balloon was advanced too far into the coronary sinus, and its excessive inflation caused rupture of the venous wall. In the other 2 patients, a tear in the great cardiac vein occurred as a consequence of a perfusion pressure that was too high; for technical reasons, the flow rate had not been accurately controlled. The three venous injuries were repaired successfully.

During none of the postmortem examinations was evidence of damage to the coronary venous system found. Likewise, in none of the 22 patients who required early reexploration for postoperative bleeding could the cause of hemorrhage be ascribed to an injury to the coronary sinus or one of its branches.

Comment

Methodology

The two major methodological limitations of the present study are readily apparent. It is a retrospective study, and it does not include a control group who received cardioplegia anterogradely. In an attempt to minimize the bias inherent to retrospective studies that extend over a long period, we analyzed the results with reference to both the entire patient population and the two surgical time frames.

As for the second concern, it must be remembered that the primary objective of this study was an assessment, on the basis of a large patient population, of the clinical relevance of coronary sinus cardioplegia in terms of feasibility, safety, and efficacy, not a comparison between two techniques of cardioplegia delivery. In this regard, the present study can be considered an extension of our initial clinical trial [7] in which the comparison of anterograde coronary perfusion with retrograde coronary sinus perfusion led to the conclusion that these two routes for cardioplegia delivery provide the same degree of intraoperative myocardial protection. In short, this study should be viewed as primarily observational, and as such, the two limitations are unlikely to bring into question the validity of its clinical implications.

Analysis of Results

FEASIBILITY. A short right atriotomy about 1 cm from and parallel to the atrioventricular groove has consistently allowed us to cannulate the coronary sinus easily and expeditiously. Occasionally, the access to the coronary sinus ostium was obstructed by an overdeveloped thebesian valve; simple retraction or even resection of the valve will facilitate catheter insertion. From this standpoint, it is noteworthy that, in adults, a single catheter size fits all coronary sinus orifices because of the compliance of the balloon.

Recently, some authors [10] have advocated blind cannulation of the coronary sinus through a pursestring suture in the right atrium to avoid the need for isolation of the right heart. Although there is no doubt about the feasibility of this approach, there are concerns as to how to accurately control both balloon positioning and balloon inflation, particularly when coronary sinus perfusion is used to deliver the initial dose of cardioplegia, as this implies transatrial cannulation of the sinus in a still-beating heart. (The issue is different if the heart has been arrested anterogradely with retrograde perfusion being used subsequently for maintaining cardioplegia, as blind intubation of the sinus is more easily accomplished in a motionless and empty heart.) In any case, additional clinical data are needed before coronary sinus cardioplegia without opening of the right atrium can be safely recommended.

SAFETY. Our 0.6% incidence of retroperfusion-related traumatic complications compares favorably with the 1% to 5% incidence of coronary complications reported after the use of direct ostial cannulation [2]. This finding supports the safety of coronary sinus cardioplegia, provided some technical precautions are taken.

The precautions include the following: (1) gentle inflation of the balloon until it occludes the lumen of the coronary sinus, but without an excessively tight fit between the catheter and the venous vessel; (2) maintenance of the catheter in the terminal portion of the coronary sinus so that the inflated balloon remains in a partially intraatrial position, thereby minimizing mechanical stress on and its attendant risk of injury to the underlying venous wall; and (3) adjustment of the flow rate of cardioplegia infusions to approximately 100 mL/min, which will prevent perfusion pressure rising above the safety threshold of 40 mm Hg. Alternatively, this variable can be monitored directly by means of the pressure line incorporated into the lumen of retroperfusion catheters. This can be of particular importance if blood cardioplegia is used because the viscoelastic properties of sanguineous perfusates are different from those of crystalloid cardioplegic solutions, and this can make more hazardous the predictability of perfusion pressure from a given flow rate. Nevertheless, our experience clearly shows that these technical measures are effective in preventing coronary venous injury, a finding that corroborates the results of almost all the clinical studies on retrograde cardioplegia through the coronary sinus [2, 11–14].

Finally, it should be noted that heart block is not a real concern even for those [11] who anchor the catheter in the sinus by means of a pursestring suture placed along the rim of the coronary sinus ostium. We think, however, that balloon inflation is a simpler means of achieving self-maintenance of the catheter in the proper position during aortic cross-clamping.

EFFICACY. As shown in Table 3, the overall mortality for patients undergoing isolated AVR was 5%, a percentage that fell to 1.5% during the last 5 years (time frame 2). These figures are within the commonly reported range [15–17]. Our findings that advanced age, urgent operation, and associated procedures increased operative mortality are also consistent with the results of other clinical series [16–20].

Data on the incidence of postoperative low output syndromes are scarce, and comparison between series is made difficult by the wide diversity in postoperative care protocols among institutions. However, our 15.2% incidence of postoperative hemodynamic failure necessitating pharmacological support is somewhat better than the 18.3% incidence reported by Christakis and associates [16] in a large series of 1,098 patients and compares very favorably with the 58% incidence in the control group in the study by Flameng and associates [21] on the effects of nifedipine (in these two studies, cardioplegia was given anterogradely). As for our 7.4% incidence of supraventricular arrhythmias postoperatively, it is in the low range compared with that in previously published reports [21, 22].

It should be stressed that the results of our study most likely reflect the level of myocardial protection that can be achieved with the use of coronary sinus cardioplegia alone, as all of our patients received cardioplegic solution only in a retrograde fashion. This contrasts with other clinical studies [13, 23, 24] in which the effects of coronary sinus perfusion are somewhat obscured by the combined use of an initial anterograde cardioplegia infusion for inducing arrest and then retrograde administration. One could argue that we also used an anterograde route for delivering our modified reperfusate. However, although we [8] have previously shown that this reperfusion solution provided additional myocardial protection, it is unlikely that if a severe ischemic insult had occurred intraoperatively, it would have been reversed only by the brief anterograde delivery of the crystalloid reperfusate at the end of the arrest period.

Thus, the overall conclusion that can be drawn from the present data is that the use of coronary sinus cardioplegia resulted in outcomes comparable with those of patients given cardioplegia in an anterograde fashion. This result supports our previous findings [7] as well as those of others [2, 12, 25] who have used retrograde coronary sinus cardioplegia for valve operations. The question can then be asked, why use a technique that appears more cumbersome than the traditional anterograde cardioplegia delivery techniques if it does not provide better protection? The reason is related to some specific advantages of the coronary sinus approach.

Advantages of Coronary Sinus Cardioplegia

Retrograde coronary sinus perfusion of cardioplegic solution in aortic valve operations offers several advantages.

1. It avoids the risk of coronary artery complications associated with direct ostial cannulation [26], in particular, that of late stenosis of the left main coronary trunk. In this setting, the coronary sinus approach is especially helpful in patients with heavily calcified aortic roots.
2. It obviates the need to interrupt the surgical procedure whenever the cardioplegic solution has to be reinfused because the catheter is self-secured in the sinus by means of balloon inflation throughout the arrest period. This eliminates the nuisance of having coronary artery perfusion cannulas intermittently obscuring the operative field and, in double-valve replacement, the time needed to reestablish exposure of the aortic valve region if cardioplegia has to be given during the mitral valve procedure.
3. It seems to retard atrial rewarming during the ischemic period [27], probably because of the topical endocardial cooling resulting from the egress of cold cardioplegic solution directly into the right cardiac chambers. This might account for the relatively low incidence of postoperative supraventricular arrhythmias in the present study.
4. It seems, at least experimentally [2], to improve the preservation of hypertrophied myocardium subjected to global ischemia. This advantage could be of special relevance to patients with left ventricular hypertrophy at the time of AVR, as the numerous venous channels in the human heart [6] are likely to behave as particularly effective conduits for delivering core-cooling and cardioplegic ingredients throughout the thickness of the myocardium [28].
5. In the laboratory setting, it is more effective than aortic root perfusion for ensuring uniform distribution of hypothermia [29–32] and cardioplegia [33] in myocardial areas distal to coronary artery obstructions. Whether this potential benefit of coronary sinus cardioplegia was of clinical importance in the present series cannot be determined, as particular referral patterns resulted in preoperative assessment of the coronary artery anatomy in only a very few of our patients. However, the clinically documented ability of retrograde coronary sinus perfusion to provide homogeneous cooling in the presence of critically obstructive multivessel coronary disease [34] lends additional support for the use of this route of cardioplegia delivery whenever AVR is performed in patients who have this pattern of coronary artery lesions.

Limitations of Coronary Sinus Cardioplegia

Like any technique, coronary sinus cardioplegia is associated with a number of drawbacks. The experience derived from the present study suggests, however, that some of them have probably been overestimated.

1. Coronary sinus injury. As already discussed, the risk is minimal provided a few simple technical precautions are taken.
2. Inadequate preservation of the right ventricle. This concern has been raised on the basis of canine studies [35]. However, the venous drainage patterns of the human right ventricle seem more suitable for retrograde perfusion [6, 33] provided the balloon catheter does not impede cardioplegia flow into the distal branches of the sinus. Thus, using radionuclide angiocardiography, we [36] have shown that right ventricular function was adequately preserved after coronary sinus cardioplegia for aortic valve operations. Similar observations have been made by others [11, 37] in patients undergoing coronary bypass procedures. These conclusions, however, pertain to the coronary sinus route, and their extrapolation to right atrial cardioplegia remains controversial [38–40].
3. Delay of cardiac arrest. There is no question that it takes a longer time to arrest the heart retrogradely than to do it through the aortic root. However, neither our results nor those of others [11, 37] suggest that this feature of coronary sinus cardioplegia has clinically detectable adverse effects on postoperative outcome, possibly because the fibrillatory state before arrest is of brief duration (on average, one to two minutes) and occurs in a cooled heart [41]. Nevertheless, except in patients who have major aortic regurgitation, the delay in arrest can be circumvented by giving the first dose of cardioplegic solution through the aortic root [13, 23, 24], a strategy that has been found highly protective in a canine model of cardioplegic arrest superimposed on acute coronary artery occlusion [10].
4. Need for bicaval cannulation. This limitation is probably the one that has made surgeons most reluctant to adopt retrograde cardioplegia through the coronary

sinus. For this reason, great interest should be paid to the development of catheter systems that can be introduced into the sinus through a stab wound in the right atrial wall and thereby allow concomitant venous drainage through a single cannula. However, until the safety and the efficacy of these new devices have been firmly established on the basis of large clinical series, we still think that bicaval cannulation is an acceptable constraint in view of the advantages that coronary sinus cardioplegia can offer in select situations.

Conclusion

In conclusion, our results support the concept that retrograde coronary sinus perfusion is a safe and effective means of cardioplegia delivery in patients undergoing AVR. Although we consider operation on the aortic valve or the aortic root as the elective situation for using coronary sinus cardioplegia, there is now sufficient experimental evidence to justify a more thorough assessment of this technique in certain subsets of patients having coronary operations, for example, those with multivessel occlusive disease [34] or those having a reoperation [14]. It is our hope that by establishing, on the basis of a large study group, the reliability of coronary sinus cardioplegia, the present study will stimulate clinical trials in these high-risk patients so that we can better define the place of the coronary sinus route in the armamentarium of cardioplegia delivery techniques.

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DISCUSSION

DR ROBERT M. MENTZER, JR (Buffalo, NY): I congratulate Professor Menasché and his colleagues on their elegant study documenting the clinical relevancy of coronary sinus cardioplegia during aortic valve operation in terms of practicality and safety.

At the State University of New York, my associates and I also have been examining the role of retrograde cardioplegia in a controlled prospective study designed to assess retrograde perfusion not only during aortic valve procedures but also during coronary bypass grafting and heart transplantation. Our study compares combined retrograde and anterograde cardioplegia with anterograde cardioplegia alone with respect to postoperative ventricular function.

To date, our experience has been substantially less than that reported by Professor Menasché, as we have studied only 70 patients. This represents just a small fraction of the total number of patients operated on in the past year by our group, that is, 70 of more than 1,400 open heart procedures. We believe this is an important 70, however, in that this group has been compared with a matched control group with respect to associated medical diseases, extent of cardiac pathology, and variables such as ejection fraction, cross-clamp time, and total bypass time.

The effect of anterograde cardioplegia and combined cardioplegia on recovery of left ventricular stroke work index for patients undergoing aortic valve and coronary bypass procedures was examined. Percent recovery of left ventricular stroke work index and the time at which the measurement was taken were considered. What you should appreciate is that in the first six hours postoperatively, there was substantial stunning of the myocardium in both groups. In contrast to the anterograde cardioplegia group, however, the combined group recovered faster and at the end of 16 hours, the left ventricular stroke work index had returned to prebypass levels.

We suggest that not only can retrograde coronary sinus cardioplegia be administered safely, but when compared with a control group, its beneficial effects can be better appreciated.

I have three questions for Professor Menasché. First, could you be a little more specific about your definition of low cardiac output syndrome, as 12% of the patients required inotropic support for more than 24 hours? Does this, in effect, represent inadequate protection?

Second, could you explain your rationale for using intermittent rather than continuous retrograde cardioplegia? We have found the latter more effective in sustaining myocardial hypothermia.

Third, would you speculate as to whether retrograde coronary sinus perfusion should become the preferred approach to preservation in patients undergoing reoperative revascularization?

DR STEVEN R. GUNDRY (Loma Linda, CA): I congratulate Dr Menasché on once again bringing to this Society results of work that he has done for many years showing the safety and effectiveness of coronary sinus cardioplegia. His pioneering efforts have stimulated a great number of us to continue our work.

He alluded to the disadvantages of retrograde cardioplegia that have kept it out of the hands of the practicing surgeon, that is, bicaval cannulation, caval snares, and a need for an atriotomy. I was frustrated by these and chose to work on transatrial intubation of the coronary sinus in a blind fashion using a flexible introducer placed in a coronary sinus cannula. This introducer, as you may know, can be shaped into a hockey-stick formation and then passed into the coronary sinus with the patient either on bypass or, as we prefer, off bypass. I will briefly update our series.

As of April, we had seen 160 consecutive patients at the University of Maryland, and at Loma Linda, we are now up to 225 consecutive patients. As of April, the breakdown was valvular disease in 44 patients and revascularization in 116, including 20 redos and 40 cases of urgent or acute myocardial infarction.

Compared with a control group of 248 patients given blood cardioplegia, our use of inotropic agents is now down to 8%. We have had no incidence of heart block and no incidence of coronary sinus damage. All patients have been able to undergo intubation of the coronary sinus, and we have not used anterograde cardioplegia in any of them.

I second Dr Menasché in his belief that this method can have wide application. The introduction of these transatrial techniques, I think, brings this important method of cardioplegia to practicing surgeons everywhere.

DR HENDRICK BARNER (St. Louis, MO): I also congratulate Dr Menasché on his pioneering efforts in retrograde coronary sinus cardioplegia. My associate, Dr Andrew Fiore, has reported the results of our randomized clinical study comparing anterograde and retrograde coronary sinus cardioplegia in which we used bicaval cannulation with a right atriotomy and found that retrograde cardioplegia was just as effective as anterograde cardioplegia.

Since the completion of that study, the transatrial catheter, which Dr Gundry described, has become available. We now use the transatrial catheter routinely for aortic valve operations with a two-stage venous cannula, for mitral valve operations using bicaval cannulation without an atriotomy, and for all repeat coronary operations using a two-stage cannula.

The major advantage of this technique is that, for valve operations, it permits the surgeon to continue working while cardioplegia is infused. For reoperations, it solves the problem of a patent internal mammary artery graft or diseased vein grafts that can embolize during anterograde infusion.

DR MENASCHÉ: Regarding Dr Mentzer's concerns, the first question was related to the definition of a low-output syndrome. As I mentioned, we considered patients who needed inotropic agents or vasopressors for more than 24 hours as having low-output syndromes. All of these patients had hemodynamic monitoring, and the definition was a conventional one, that is, a cardiac index lower than 2 $L/min/m^2$ in spite of adequate volume loading and correction of possible arrhythmias or metabolic disturbances.

There was no real rationale for using intermittent rather than continuous retrograde cardioplegia. When we started in 1980 and wanted to use this technique, we were a little concerned about the possibility of inducing some form of myocardial edema, and this was why we chose to perfuse the cardioplegic solution in an intermittent fashion. However, assuming that the first infusion takes approximately ten minutes and the other infusions five to seven minutes, we may end up with an almost continuous infusion of cardioplegic solution.

Regarding the redo procedures, I really believe that retrograde cardioplegia is a very nice technique for use in this setting for at least two reasons. Usually the native coronary arteries of these patients are occluded, and it gives us a chance to perfuse cardioplegia retrogradely. The anterograde approach carries the risk of distal embolization of atheromatous material that can be dislodged from still patent saphenous vein grafts.

Dr Gundry, I acknowledge your pioneering efforts in developing this blind approach for cannulating the coronary sinus and congratulate you for your excellent clinical results. They actually support the safety and the effectiveness of the retrograde approach.

Finally, I thank Dr Barner for his comments. I think the report he and his group have published as well as their ongoing experience bring further clinical evidence that this technique is safe and effective and that in select patients, it can really be useful and should now be part of our armamentarium of cardioplegia delivery techniques.

Skeletonization of the Atrioventricular Node for AV Node Reentrant Tachycardia: Experience With 32 Patients

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We describe our experience with operative therapy for atrioventricular (AV) node tachycardia using an anatomically guided procedure. The operative rationale was to dissect the AV node from most of its atrial inputs (AV node "skeletonization") with the intent of altering the perinodal substrate and preventing reentry. The anteroseptal and posteroseptal regions were initially approached epicardially to facilitate identification of anatomical structures. Under normothermic cardiopulmonary bypass, the right atrial septum was mobilized and the intermediate AV node was exposed anterior to the tendon of Todaro. Atrioventricular node conduction was monitored electrocardiographically throughout the procedure. Ablation of concomitant accessory pathways was done prior to AV node skeletonization. Thirty-two patients aged 9 to 67 years (mean age, 30 years) underwent operation. Five patients had concomitant accessory pathways in addition to AV node reentry. At electrophysiological study before discharge, no patient had AV block although anterograde and retrograde Wenckebach cycle lengths were significantly prolonged. Six patients had retrograde AV block. Twenty-nine patients are free from arrhythmia and require no antiarrhythmic medication after a follow-up of 1 month to 45 months (mean follow-up, 17 months). Three patients had recurrence of tachycardia ten days, 2 months, and 7 months postoperatively. All patients subsequently had a successful reoperation.

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Until recently, His bundle ablation was the only electrophysiological intervention for patients with atrioventricular (AV) node reentrant tachycardia [1, 2]. The fortuitous cure of a patient with AV node reentrant tachycardia after attempted surgical ablation by Pritchett and colleagues [3] demonstrated that direct ablative surgical intervention for AV node reentrant tachycardia with preservation of AV node conduction was feasible. Subsequently, two routes of research were explored: (1) permanent cryosurgical modification of the perinodal area [4, 5] or AV node [6, 7] and (2) sharp dissection of the AV node with [8] or without [9] myocardial mapping.

We report our experience with AV node "skeletonization" in 33 patients. The intermediate AV node is dissected and exposed using sharp dissection guided solely by anatomical landmarks.

Material and Methods

Surgical Gross Anatomy of Atrioventricular Node

Although there are marked discrepancies between the definition of the AV node as given by morphologists, clinicians, and electrophysiologists [10], the following gross anatomical description of this node is consistent with the original description by Tawara [11], the European group [12], and our macroscopic dissection. The AV node comprises (1) the compact node within the atrial membranous septum, (2) the penetrating node where it enters the right fibrous trigone (central fibrous body of pathologists [13]), (3) the intermediate AV node, and (4) the atrial inputs to the AV node. Although the entire AV node can be macroscopically dissected in a heart specimen (Fig 1), current surgical techniques permit dissection and exposure of only the intermediate node and its atrial inputs.

INTERMEDIATE ATRIOVENTRICULAR NODE. The intermediate AV node is a triangular bundle of pale myocardial fibers (lighter in color than surrounding atrial myocardium). The apex of the triangle is at the posteroinferior angle of the atrial membranous septum (penetrating bundle). The anterior side is vertical and slightly posterior to the posterosuperior process of the left ventricle, namely, the mitral valve annulus. The posterior side is parallel to the tendon of Todaro. The base is ill defined in the region of the coronary sinus os where the intermediate AV node transforms into common myocardium. The intermediate AV node is covered by the inferomedial right atrial wall.

Mobilization of the right atrial wall with subsequent exposure of the intermediate AV node is feasible because there is a plane of cleavage between the AV node and the right atrial wall. After exposure, the intermediate AV node is identified by its oblique, lightly colored myocardial fibers with fatty streaks converging into the atrial membranous septum. The intermediate AV node is not encased in a fibrotic sheath, and its limits are not well

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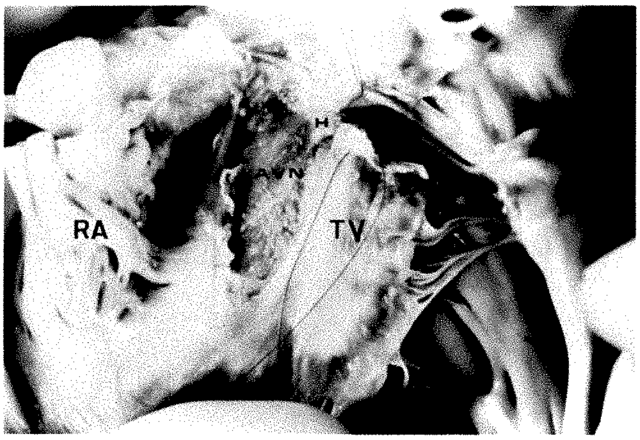

*Fig 1. View of atrioventricular (AV) node dissection in a heart specimen. The right atrial wall (*RA*) has been resected to expose the intermediate AV node (*AVN*). The tricuspid valve (*TV*) and the membranous septum have been opened to expose the compact node and His bundle (*H*).*

demarcated, particularly at the attachment between the intermediate AV node and the atria (atrial inputs), as originally well described by Tawara [11].

ATRIAL INPUTS. The intermediate AV node, at its atrial segment, is distributed into three well-defined groups of nodal cells labeled as deep, superficial, and posterior [12]. The deep group lies left of the tendon of Todaro and is in contact with the left atrial wall, near the apex of the intermediate AV node. The superficial group lies right of the tendon of Todaro and is in contact with the right atrial septal myocardium anterior to the fossa ovalis. The posterior group is in contact with a myocardial bundle that can be traced from the coronary sinus os. These three nodal groups and their atrial myocardium connection define three homologous deep, superficial, and posterior atrial inputs.

ANATOMICAL LANDMARKS. Anatomical landmarks are (1) the atrial membranous septum, identified as a whitish membrane below the aortic root, (2) the posterosuperior process of the left ventricle, (3) the tendon of Todaro, and (4) the coronary sinus orifice (Fig 2). These landmarks circumscribe a triangle that occupies only a segment of the triangle of Koch.

ATRIOVENTRICULAR NODE AREA CONCEPT. The AV node area concept is based on the absence of well-delineated anatomical limits. Consequently, the anatomist is compelled to draw an "invisible barrier" that encompasses the AV node and its inputs as identified by the histologist.

Clinical Material

Between December 1985 and July 1989, 32 patients underwent AV node skeletonization for AV node reentrant tachycardia. There were 29 female and 3 male patients. Age ranged from 9 to 67 years (mean age, 30 years). All patients had recurrent paroxysmal tachycardia associated with palpitations. Nine patients had associated syncope, light-headedness, or dizziness. Duration of symptoms ranged from 2 to 36 years (mean duration, 10 years). No patient had associated cardiac disease as documented by routine echocardiographic Doppler study in all and left heart catheterization in patients more than 40 years of age. In patient 15, catheter ablation of the His bundle had been attempted at another institution.

Preoperative Electrophysiological Studies

The main electrophysiological characteristics are summarized in Table 1. The diagnosis of AV node reentrant tachycardia was based on one or more of the following characteristics [14]: (1) presence of anterograde or retrograde AV node pathway physiology; (2) initiation of the tachycardia following a critical gap in A-H interval prolongation; (3) retrograde atrial activation pattern during tachycardia similar to AV node activation pattern with earliest activation recorded at the His bundle catheter or proximal coronary sinus catheter; (4) failure of ventricular extrastimuli to advance atrial activation during tachycardia; and (5) shortest ventriculoatrial interval less than 60 ms.

Twenty-six patients had anterograde dual AV node pathway physiology, and 7 of them had associated retrograde dual pathway physiology. Two patients had only retrograde dual AV node pathway physiology, and 3 patients had neither anterograde nor retrograde dual pathway physiology. The data were not available for 1 patient.

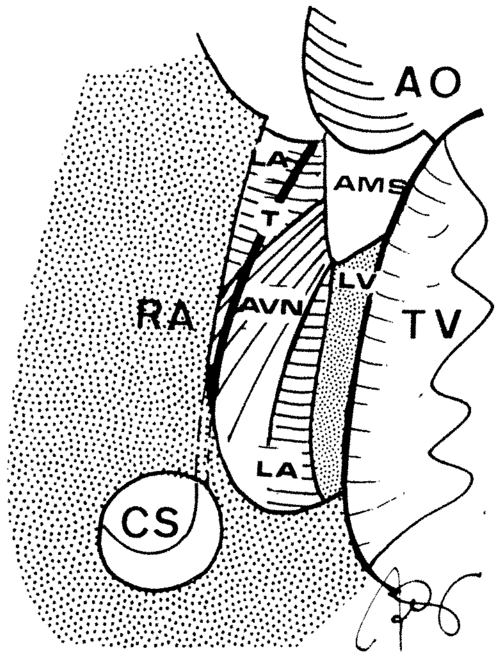

*Fig 2. Anatomical landmarks of the intermediate atrioventricular node (*AVN*) in a view similar to that in Figure 1. The right atrial wall (*RA*) has been removed in the septal area. (*AMS* = atrial membranous septum; *AO* = aortic root; *CS* = coronary sinus; *LA* = left atrium; *LV* = posterosuperior process of left ventricle; *T* = tendon of Todaro; *TV* = tricuspid valve.)*

Table 1. Preoperative Electrophysiological Data[a]

			Wenckebach Cycle Length		Dual AV Node Pathway Physiology			Tachycardia					
Patient No.	Sinus Node Cycle Length	A-H Interval	Antero-grade	Retro-grade	Antero-grade	Retro-grade	Bidirec-tional	Common Form[b]	Uncommon Form	Cycle Length	Shortest VA Duration	Site[c]	Miscellaneous
1	680	50	260	260	Yes	No	No	Yes	. . .	320	40	HB	. . .
2	830	55	240	240	Yes	No	. . .	Yes	No	240	50	HB	. . .
3	680	80	290	330	Yes	No	No	Yes	No	340	35	HB	. . .
4	810	65	340	440	Yes	No	No	Yes	No	360	10	HB	. . .
5	400	50	230	360	No	No	No	Yes	No	350	20	CS	. . .
6	500	80	350	270	Yes	No	No	No	Yes	320	140	HB	. . .
7	770	50	390	280	Yes	No	No	No	Yes	350	245	CS	Ablation of left ventricular free wall accessory pathway 11 mo previously
8	640	55	260	320	Yes	No	No	Yes	No	260	40	CS	Mahaim fibers
9	820	45	300	300	Yes	No	No	Yes	No	340	0	HB	. . .
10	870	120	330	440	No	Yes	No	No	Yes	400	110	HB	. . .
11	940	70	410	550	Yes	Yes	Yes	Yes	No	210–280	70–90	HB	. . .
12	660	70	260	250	Yes	No	No	Yes	No	270	0	HB	. . .
13	630	60	270	270	Yes	Yes	Yes	Yes	No	270	20	HB	. . .
14	720	70	540	440	Yes	Yes	Yes	No	Yes	500	70	HB	. . .
15	650	70	330	360	Yes	No	No	Yes	. . .	205	20	HB	Attempted His bundle ablation previously
16	690	60	290	210	Yes	No	No	Yes	. . .	340	30	CS	. . .
17	660–730	80	410	420	Yes	Yes	Yes	Yes	. . .	270	40	HB	. . .
18	1,000	35	<250	<250	Yes	No	No	Yes	No	250	50	HB	Mahaim fibers
19	550	80	260	230	Yes	Yes	Yes	Yes	. . .	240	−10	HB	. . .
20	1,060	60	310	. . .	Yes	No	No	Yes	. . .	290	50	HB	. . .
21	720	75	270	340	Yes	Yes	Yes	Yes	No	290	50	HB	. . .
22	625	30	<190	<250	No	No	No	Yes	. . .	250	50	HB	Associated posteroseptal accessory pathway
23	780	65	330–540	290	Yes	No	No	Yes	No	390	10	HB	. . .
24	810	80	330	. . .	Yes	No	No	Yes	No	315	−25	HB	. . .
25	610	50	390	Block	No	No	No	Yes	No	330–410	<60	HB-CS	. . .
26	650	55	<250	<250	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	Posteroseptal AV node reentrant tachycardia
27	900	75	370	390	No	Yes	No	No	Yes	420–630	75	HB	Associated posteroseptal accessory pathway
28	600	80	270	270	Yes	No	No	Yes	No	280–380	30	HB	. . .
29	835	80	. . .	. . .	Yes	Yes	Yes	Yes	No	430	80	HB	. . .
30	770	110	370–500	<440	Yes	No	No	Yes	No	345	70	HB	. . .
31	760	80	300	300	Yes	No	No	Yes	No	320	30	HB	. . .
32	760	90	<400	<250	Yes	No	No	Yes	No	310	0	HB	. . .

[a] Time is shown in milliseconds. [b] This means slow-fast. [c] This is the site of the shortest VA interval during tachycardia.

AV = atrioventricular; CS = coronary sinus os catheter; HB = His bundle catheter; VA = ventriculoatrial.

Twenty-six patients had the common form of tachycardia (slow-fast) with short ventriculoatrial intervals. Five patients had the uncommon form of AV node reentrant tachycardia with long ventriculoatrial intervals. In 1 patient, tachycardia could not be induced in the laboratory. Twenty-six patients had earliest atrial activation during tachycardia at the His bundle catheter alone. Five patients had associated tachycardia with preexcitation syndrome.

In Patient 7, a left ventricular free wall accessory pathway was surgically ablated 11 months before AV node skeletonization because AV node reentrant tachycardia became manifest only after ablation of the accessory AV pathway. Patients 22 and 26 had associated posteroseptal accessory AV pathways. Patients 8 and 17 had nodoventricular Mahaim fibers associated with a right ventricular pathway with AV node–His bundle physiology [15].

Operative Technique

The heart was exposed through a median sternotomy in all patients. Anterograde and retrograde AV node conduction was monitored in all patients during dissection of the AV node region. Dissection was momentarily interrupted when anterograde block occurred. Atrial septal endocardial mapping using a single exploring bipolar electrode or a 60-electrode plaque with simultaneous recordings was obtained in patients but was not used to either localize the AV node–His bundle or guide the surgical technique.

Atrioventricular node skeletonization was carried out on the normothermic, beating heart according to the following steps. (1) Before heparinization, anteroseptal and posteroseptal regions were approached to facilitate the endocardial dissection by identifying anatomical structures. (2) Under normothermic cardiopulmonary bypass, the atrial septum was approached anterior to the tendon of Todaro, and the intermediate AV node was exposed.

The anteroseptal dissection was identical to the one described for anteroseptal accessory AV pathways [16]. The AV fat pad in the right coronary fossa was mobilized en bloc. The sinus node artery was identified and spared. The tricuspid valve annulus was exposed up to its septal attachment.

The posteroseptal dissection was aimed at separating the inferomedial right atrial wall from the posterosuperior process of the left ventricle. A right posteroseptal dissection was carried out as previously described [17]. The inferior right ventricular fat pad and the right atrial left ventricular fat pad were mobilized en bloc. The middle cardiac vein was spared. The inferomedial right atrial wall and the posterosuperior process of the left ventricle were exposed. Special attention was given to hemostasis.

Cardiopulmonary bypass was instituted using routine aortic cannulation, while double venous cannulation was used. The right atrial pursestrings were placed posteriorly near the homologous orifice of the vena cava. After the inception of normothermic cardiopulmonary bypass, the heart was electrically fibrillated. The superior vena cava and the inferior vena cava were snared with tapes. A right atriotomy was performed along the exposed tricuspid annulus in the anteroseptal region and was extended along the AV fat pad anteriorly. Stay sutures were used to expose the right atrium. The atrial septum was carefully inspected for defects, and a patent foramen ovale, if present, was closed. The heart was defibrillated.

For intermediate AV node skeletonization, the right atriotomy along the tricuspid annulus was extended up to the atrial membranous septum. The right atrial wall was dissected and separated from the aortic root and the adjacent left atrial wall. The superior attachment of the tendon of Todaro was identified. The endocardium of the inferomedial right atrial wall was incised along the muscular segment of the septal portion of the tricuspid annulus. The already dissected posteroseptal space was entered.

The posteroseptal atrial incision was extended up to the atrial membranous septum. The dissection of the right atrial wall was completed and extended inferiorly toward the coronary sinus orifice and superiorly where a plane of cleavage was identified between the adjacent right atrial wall and the underlying intermediate AV node. The dissection was extended to meet the anteroseptal dissection and the already exposed plane of dissection between the right and left atrial walls.

The septal atrial wall was separated from its attachment to the atrial membranous septum and deflected posteriorly to expose the AV node. The dissection of the septal atrial wall was completed to expose the entire tendon of Todaro. At this point, the intermediate AV node was exposed and identified by its oblique, pale myocardial fibers and was carefully dissected and freed from surrounding tissue on its anterior and posterior sides and its right aspect (Fig 3).

After completion of AV node skeletonization, the superficial and posterior atrial inputs to the AV node were separated. Only the deep inputs were left intact.

The atriotomies were closed by reattaching the atrial wall to the tricuspid annulus using 4-0 Prolene (Ethicon, Somerville, NJ) running sutures. Cardiopulmonary bypass was discontinued. The mean cardiopulmonary bypass time was 56 minutes (range, 25 to 88 minutes).

Associated Arrhythmias

Associated arrhythmias were ablated before AV node skeletonization. The two posteroseptal pathways were ablated using an epicardial approach on the beating heart before institution of cardiopulmonary bypass [17]. The two Mahaim fibers were ablated using a right ventricular free wall epicardial dissection [15], and a subsequent right atriotomy along the tricuspid annulus and concomitant right atrial resection in the region of insertion of the Mahaim fiber for histological examination were performed [18].

Before the closing, pairs of temporary pacing electrodes were attached to each chamber of the heart for postoperative electrophysiological studies, which were conducted ten to 15 days postoperatively. Pharmacological testing or intracardiac catheter recording or both were used in select patients.

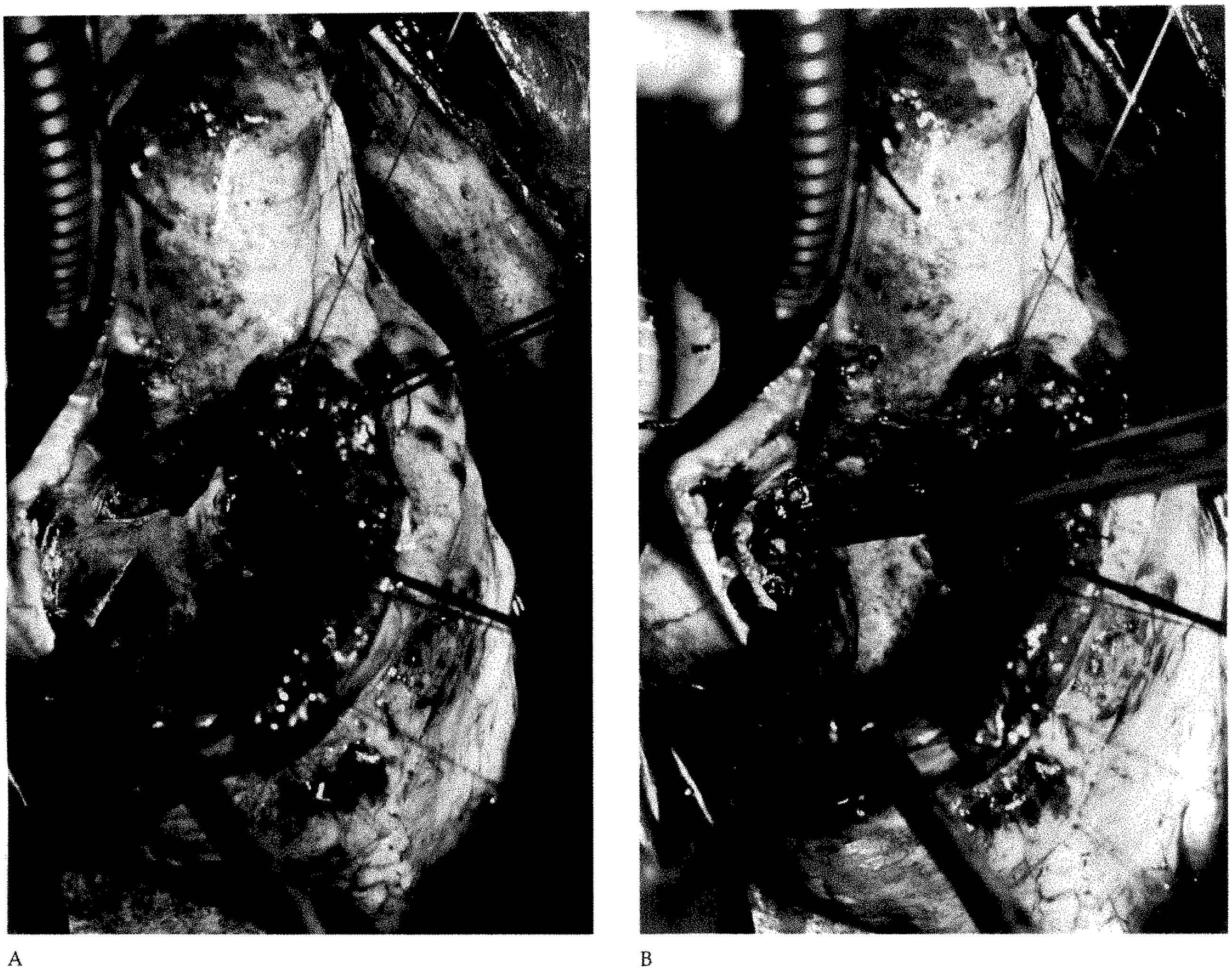

Fig 3. Operative views. The atrial septal region is exposed. (A) The anteroseptal region and the posteroseptal region have been dissected. A strip of right atrial wall is still intact and covers the intermediate AV node. (B) The right atrial septal wall has been mobilized and the intermediate AV node is exposed (pointed out by tissue forceps).

Results

Surgical Risk

There were no surgical complications intraoperatively or postoperatively. There was no permanent complete heart block. Two patients (patient 9 and patient 17) had second-degree heart block with Wenckebach periods. The 2 patients were asymptomatic at rest and during exercise with an adequate heart rate response during treadmill testing.

Efficacy for Arrhythmia

Anterograde Wenckebach cycle length was significantly prolonged from a mean of 326 ± 78 ms preoperatively to a mean of 404 ± 89 ms postoperatively ($p = 0.001$). As mentioned, 2 patients had second-degree heart block. Retrograde Wenckebach cycle length was significantly prolonged from a mean of 318 ± 8 ms preoperatively to a mean of 377 ± 128 ms postoperatively. Six patients had complete retrograde AV block. Anterograde dual AV node pathway physiology was present postoperatively in 4 patients, 2 of whom had associated inducible AV node reentrant tachycardia, which was nonsustained in patient 2 and sustained in patient 7 (Table 2).

Long-Term Follow-up

Three patients had recurrence of tachycardia. Patient 2 with inducible nonsustained tachycardia ten days postoperatively had clinical recurrence of tachycardia 7 months postoperatively. Patient 7, who had early recurrence, continued to have tachycardia. Patient 21 had recurrence of tachycardia 2 months after operation, although she had a normal electrophysiological study postoperatively with a retrograde Wenckebach cycle length of 330 ms. Two of the 4 patients with anterograde dual AV node pathway physiology had no clinical recurrence of tachycardia after 3 years' and 16 months' follow-up.

The 3 patients with recurrent tachycardia were reoperated on 11, 11, and 9 months after the initial operation. At reoperation, a thin layer of whitish endocardial fibrosis was found covering the atrial septum. Anatomical landmarks could be identified, and AV node skeletonization was carried out without complication.

Table 2. Postoperative Electrophysiological Data[a]

Patient No.	Sinus Node Cycle Length	A-H Interval	Wenckebach Cycle Length		Dual AV Node Pathway Physiology			Tachycardia					Miscellaneous
			Antero-grade	Retro-grade	Antero-grade	Retro-grade	Bidirectional	Common Form[b]	Uncommon Form	Cycle Length	Shortest VA Duration	Site[c]	
1	. . .	. . .	350	750	No	No	No	. . .	. . .	. . .	. . .	. . .	. . .
2	590	55	320–330	280	Yes	No	No	Yes	No	310	50	HB	Nonsustained reentrant tachycardia
3	660	50	410	530	Yes	No	No	. . .	. . .	. . .	. . .	. . .	. . .
4	730	65	500	350	No	No	No	. . .	. . .	. . .	. . .	. . .	. . .
5	570	90	450	Block	No	No	No	. . .	. . .	. . .	. . .	. . .	. . .
6	640	50	330	380	No	No	No	. . .	. . .	. . .	. . .	. . .	. . .
7	860	40	310	370	Yes	Yes	Yes	No	Yes	450	30–40	HB	Sustained reentrant tachycardia
8	640	70	400	440	No	No	No	. . .	. . .	. . .	. . .	. . .	. . .
9	680	130	620	Block	No	No	No	. . .	. . .	. . .	. . .	. . .	. . .
10	635	150	400	Block	No	No	No	. . .	. . .	. . .	. . .	. . .	. . .
11	1,150	50	380	300	No	No	No	. . .	. . .	. . .	. . .	. . .	. . .
12	620	60	360	300	No	No	No	. . .	. . .	. . .	. . .	. . .	. . .
13	540	60	310	350	No	No	No	. . .	. . .	. . .	. . .	. . .	. . .
14	890	85	Wck	830	No	No	No	. . .	. . .	. . .	. . .	. . .	. . .
15	560	70	260	330	No	No	No	. . .	. . .	. . .	. . .	. . .	. . .
16	850	50	390	270	No	No	No	. . .	. . .	. . .	. . .	. . .	. . .
17	850	65	390	260	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .
18	1,000	60	440	310	No	No	No	. . .	. . .	. . .	. . .	. . .	. . .
19	510	70	Wck	500	No	No	No	. . .	. . .	. . .	. . .	. . .	. . .
20	770	110	450	310	Yes	No	No	. . .	. . .	. . .	. . .	. . .	. . .
21	820	70	470	330	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .
22	640	55	420	Block	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .
23	650	75	550	330	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .
24	580	70	330	Block	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .
25	610	50	450	Block	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .
26	660	. . .	370	440	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .
27	860	. . .	620	700	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .
28	610	40	360	320	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .
29	920	. . .	400	550	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .
30	520	. . .	440	360	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .
31	650–670	. . .	<280	<270	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .
32	600	. . .	470	<250	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .

[a] Time is shown in milliseconds. [b] This means slow-fast. [c] This is the site of the shortest VA interval during tachycardia.

AV = atrioventricular; HB = His bundle catheter; VA = ventriculoatrial; Wck = Wenckebach block.

Patient 2 underwent reoperation after 11 months. The initial operation was considered incomplete because of insufficient posteroseptal dissection and intact atrial septum. She underwent complete AV node skeletonization at the second operation. Patient 7 with early recurrence had repeat AV node skeletonization, although he had apparently had a satisfactory skeletonization at the first operation 11 months before. Patient 21 had a similar repeat AV node skeletonization after an apparently satisfactory dissection 9 months before. The second operation was uncomplicated in all patients. Patient 21 has second-degree heart block with an adequate heart rate response during exercise.

In total, all patients are free from AV node tachycardia and no longer require antiarrhythmic drug therapy after a mean follow-up of 17 months (range, 1 to 45 months).

Comment

Surgical Risk

There were no complications. There was no permanent complete heart block. No patient required pacemaker implantation. The 3 patients with second-degree block were asymptomatic. Consequently, the primary objective of designing a low-risk surgical alternative to His bundle ablation was achieved.

Surgical Efficacy

In our initial experience, a second operation was required in 3 patients to achieve uniform success. Potential markers for long-term success based on postoperative electrophysiological study are absence of dual AV node pathway physiology and presence of retrograde AV block. The absence of discrete anatomical end points may explain failures that can be corrected at a second operation.

Johnson and colleagues [19, 20] have reported the largest world series of 69 patients using sharp dissection of the AV node area on the cardioplegia arrested heart. There were no long-term surgical complications. Two patients had early recurrence, and 5 had late relapses, 3 of whom had a second operation. Two patients had pacemaker implantation for permanent complete heart block. Cox and associates [21, 22] have recently reported uniform success in a series of 23 patients using perinodal cryoablation. Surgical efficacy has established the direct surgical approach as an alternative electrophysiological intervention to His bundle ablation. However, recently Haissaguerre and co-workers [23] described a nonoperative alternative in 21 patients managed with AV node alteration using catheter ablation. Sixteen patients were free from arrhythmia without antiarrhythmic drug therapy after a mean follow-up of 14 months (range, 7 to 42 months). Two patients had permanent heart block.

The surgical rationale is based on ablation of a discrete arrhythmogenic anatomical substrate guided by electrophysiological cardiac mapping with restoration of normal function. The paradigm is surgical ablation of the accessory AV pathway in Wolff-Parkinson-White syndrome. However, such a rationale cannot apply to AV node reentrant tachycardia because a critical segment of the circuit has not been anatomically documented and AV node mapping is technically difficult and not uniformly possible. Clinical electrophysiological studies document the site of the entire AV node reentry to be confined to the AV node with atrial and ventricular output similar in both tachycardia and normal AV conduction, on the following evidence:

1. Atrial and ventricular activity can be transiently dissociated from the tachycardia without affecting the tachycardia cycle length, suggesting that neither atrium nor ventricle is an obligatory segment of the reentrant circuit [24].
2. Programmed stimulation can delay, on occasion, the following tachycardia cycle, suggesting that part of the tachycardia circuit is penetrated by concealed conduction [25].
3. Retrograde Wenckebach cycle length during pacing is longer than the tachycardia cycle length, and A-H and H-A intervals during pacing are longer than during tachycardia, suggesting the presence of an upper (ventricular) and lower (atrial) common pathway [26].

However, the presence of atrial or ventricular dissociation, or both, from the tachycardia circuit does not rule out the possibility that a perinodal cuff of atrial myocardium cannot be captured by an atrial extrastimuli but can participate in the AV node circuit. Consequently, the role of atrial inputs to the AV node cannot be ruled out.

Experimental studies, primarily conducted on the rabbit heart, have documented that AV node reentry can be confined to the AV node [27, 28] or that the AV node circuit can involve the AV node and its atrial input [29, 30]. These latter experiments support the concept of the AV node area including the AV node and surrounding atrial myocardium, and reconcile the definition of AV node area based on gross anatomy with the one based on functional criteria.

Despite compelling electrophysiological evidence of functional "longitudinal" AV node dissociation, there is no pathological evidence of anatomical dissociation in the form of either intranodal partition or "extranodal" pathways [31].

In summary, operation for reentrant AV node tachycardia cannot be aimed at a discrete anatomical substrate identified by cardiac mapping. Our current approach is based on the concept of a "greater" AV node area including the AV node as defined by histologists, the atrial-nodal junction (atrial inputs), and surrounding atrial myocardium. It is speculated that the integrity of the entire AV node area is necessary for AV node tachycardia to occur and that ablation of posterior and superficial atrial inputs as well as surrounding right atrial wall (AV node skeletonization) will prevent AV node reentrant tachycardia. Atrioventricular node dissection is associated with side effects such as AV node denervation or direct injury, which might, per se, prevent tachycardias. Consequently, successful surgical intervention is not compelling evidence of valid surgical rationale. All current surgical approaches are based on the same rationale and aimed at the "perinodal" zone using either sharp dissection [9] or cryoablation [21].

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DISCUSSION

DR JAMES L. COX (St. Louis, MO): Dr Guiraudon and his colleagues are to be congratulated on their efforts to cure AV node reentrant tachycardia by surgical means and on their many contributions in other areas of cardiac arrhythmia surgery. The paper presented today represents the third large series reported in which patients with AV node reentrant tachycardia have been treated surgically.

A surgical dissection technique for the treatment of AV node reentrant tachycardia similar to the one employed by Dr Guiraudon and his colleagues was first described by Drs Ross and Johnson from Westmead Hospital in Sydney, Australia, in 1985. Last month, Dr Johnson published his results with 69 patients, and they were quite similar to those reported here today.

At our institution, we use a 3-mm cryoprobe to selectively ablate the tissues around the AV node in these patients rather than perform a surgical dissection.

We reported our results in 23 patients at the American Association for Thoracic Surgery meeting earlier this year. There were no operative deaths, no failures, no instances of heart block, no recurrences, and no reoperations, and the follow-up is now 7 years. Several other surgeons around the United States have also employed this simple procedure with identical results, so we are now aware of 72 patients who have undergone the cryosurgical procedure with uniform success.

In comparing the results of the two techniques, the surgical dissection technique has a failure rate of 4% to 9%, an incidence of heart block of 3% to 6%, and the need for a second operation to attain surgical success in about 10% of the patients. I have several questions for Dr Guiraudon. First, because you are a well-known advocate of the use of cryosurgery for other types of cardiac arrhythmias, why, in view of these results, have you not yet switched to the perinodal cryosurgical technique?

Second, the manuscript refers to 32 patients. In your discussion of our paper at the American Association for Thoracic Surgery meeting in May, you mentioned 41 patients who had undergone this type of operation. Could you please clarify the numbers?

Third, because the substrate for AV node reentrant tachycardia is dual AV conduction pathways, do you consider the 4 patients in your series with persistent dual AV node pathways postoperatively to represent failures?

Fourth, 2 patients in your series had second-degree heart block and Wenckebach periodicity and therefore are likely to need pacemakers at some time in the future. Do you consider that to be a major complication?

Finally, the group in Australia updated its surgical dissection series to 106 patients at a meeting 3 weeks ago, and David Johnson made the statement that their results were getting worse with experience, not better. Has that been your observation as well?

DR GUIRAUDON: Thank you very much, Dr Cox. We have studied and advocated cryosurgical modification of the AV node. However, we were skeptical about the reproducibility and reliability of the technique and therefore preferred sharp dissection based on well-defined anatomical landmarks. Your own results using perinodal cryoablation are compelling arguments that cryosurgery is reliable and efficient in that setting.

When I discussed your paper at the meeting of the American Association for Thoracic Surgery, the 41 patients I mentioned included patients I have operated on while visiting Holland, France, and the United States. Today, I present our series of 32 patients in London.

Does surgical efficacy decrease with the surgeon's experience, as David Johnson might have suggested? I guess that increased experience should be associated with a lower risk of permanent heart block, undue injury to the skeletonized AV node, and discrete ablation of the posterior and superficial atrial inputs. Consequently, if the surgical rationale is correct, the overall results would improve in terms of efficacy and surgical risk.

Our 2 patients with postoperative Wenckebach block are asymptomatic and have a normal 1:1 ventricular response during exercise. Because the conduction defect is proximal (A-H prolongation), it is not anticipated that they will ever need to have a permanent pacemaker implanted.

Effect of Omental, Intercostal, and Internal Mammary Artery Pedicle Wraps on Bronchial Healing

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Keith E. McEwen, MD, Philip R. Faught, MD, Michael E. Miller, PhD,
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Bronchial transection and devascularization is necessary in the course of sleeve resection or lung transplantation, leaving distal bronchial segments ischemic and subject to stricture or dehiscence. Thirty mongrel dogs underwent left lung autotransplantation. The bronchial anastomosis was wrapped with omentum (n = 9), intercostal muscle pedicle (n = 9), or internal mammary artery pedicle grafts (n = 6). Six control animals underwent bronchial anastomosis without an external wrap. Bronchial revascularization by capillary ingrowth from the pedicle to the bronchial submucosal plexus was demonstrated with all three types of vascular pedicle grafts; however, more consistent and confluent vascular ingrowth was provided by internal mammary artery pedicle grafts. Additionally, the bronchial anastomotic cross-sectional area was significantly better in the internal mammary artery group (84.5 ± 3.3) as compared with that of the omental (68.4 ± 8.3), intercostal muscle (66.9 ± 10.9), or control groups (70.2 ± 7.6). An internal mammary artery pedicle graft and the presence of dense confluent submucosal vascular ingrowth from any pedicle graft were independently predictive ($p < 0.05$) of minimizing bronchial anastomotic narrowing. These data are consistent with previous findings suggesting that omental and intercostal muscle pedicle grafts promote early bronchial revascularization; moreover, the data demonstrate the superiority of an internal mammary artery pedicle graft to provide submucosal vascular ingrowth and to minimize anastomotic stenosis.

(Ann Thorac Surg 1990;49:574–9)

The bronchial circulation accepts only 1% of the total cardiac output. It is, however, critical in maintaining the integrity of the bronchial wall and mucosa [1]. Radical bronchial dissection with bronchial arterial disruption is often necessary in the course of sleeve resections, repair of traumatic tracheobronchial injuries, and lung transplantations. This leaves the distal bronchial segment relatively ischemic, thus subject to mucosal necrosis, luminal stricture, or even frank dehiscence. Spontaneous revascularization of the distal bronchial circulation has been shown to occur between 15 and 30 postoperative days [2–5]. It is generally believed, however, that earlier revascularization will promote uncomplicated bronchial healing [5].

Various techniques have been described to protect bronchial suture lines and anastomoses. These include pleural or pericardial flaps [6, 7], telescoping of the distal bronchial end into the proximal segment [8], and transection of the bronchus close to the pulmonary hilus [9]. These techniques, although mechanically supporting bronchial suture lines, have not been found to provide early revascularization to ischemic tissue. In contrast, vascularized pedicle grafts have been shown to provide a source of rapid capillary ingrowth into ischemic bronchial tissue. Cooper and associates [5] have demonstrated initial mucosal vascular communications when canine bronchial suture lines were wrapped with greater omentum. These experimental results using the greater omentum are in part responsible for the recent success of human lung allografts. Intercostal muscle grafts have also been used as a source of capillary ingrowth [10, 11]. This study investigates the use of an internal mammary artery pedicle graft as a source for indirectly revascularizing ischemic bronchial segments as compared with previously described omental and intercostal muscle pedicle grafts.

Material and Methods

Thirty conditioned mongrel dogs weighing from 18 to 23 kg were selected for this study. All animals were humanely cared for in accordance with the "Principles of Laboratory Animal Care" and the "Guide for the Care and Use of Laboratory Animals" (NIH Publication No. 85-23, revised 1985). The dogs were anesthetized and maintained on a combination of halothane and oxygen.

Thirty dogs received an internal mammary artery (IMA, n = 6), omental (OM, n = 9), intercostal muscle (ICM, n = 9), or no pedicle wrap (control, n = 6) around the ischemic "donor" bronchus after single-lung autotransplantation. All vascular pedicle grafts were mobilized before pneumonectomy. After upper midline laparotomy,

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the greater omentum, based on the right gastroepiploic artery, was advanced through a small anterior incision in the left hemidiaphragm beneath the xiphoid process. Both IMA and ICM pedicle grafts were directly mobilized through a left posterolateral thoracotomy in the fifth intercostal space. The ICM pedicle was based on the fifth intercostal artery and vein. The IMA and vein, along with a 1-cm segment of intrathoracic muscle medial and lateral to the vessels, were dissected from the chest wall using electrocautery beginning at their origin and extending to the terminal bifurcation (Fig 1A).

Left lung autotransplantation was performed using a modification of the technique as described by Cooper and associates [5]. After dissection and isolation of the pulmonary artery and vein, as well as the left main bronchus, an 8F balloon-tip catheter (American Edwards Laboratories, Santa Ana, CA) was used to occlude the left main bronchus. Left pneumonectomy was accomplished by dividing the pulmonary artery and the left main bronchus at their respective midpoints. The pulmonary veins were divided and left with a small cuff of left atrium. The left atrial cuff was then anastomosed with a running 5-0 Prolene suture (Ethicon, Somerville, NJ) using a horizontal mattress suture technique. The bronchial anastomosis was carried out with interrupted horizontal mattress sutures of 5-0 Vicryl (Ethicon) and the bronchial occluding catheter was removed. The pulmonary artery anastomosis was performed with a continuous running 6-0 Prolene suture. Before reestablishing pulmonary artery flow, retrograde flushing was accomplished by brief release of the left atrial clamp. Except for the control animals, the bronchial anastomosis was then circumferentially wrapped with the OM or with the pleural surface of either the ICM or distal IMA pedicle grafts (Fig 1B). The surfaces of the pedicle grafts were closely coapted and gently sutured to the bronchial adventitia with interrupted 5-0 Vicryl sutures in four-quadrant fashion while avoiding tension on the pedicle vascular supply. The animals were maintained on prophylactic antibiotics consisting of 1 g of intravenous Kefzol (Eli Lilly, Indianapolis, IN) intraoperatively, followed by 1.2×10^7 U of intramuscular Distrycillin (Solvay Veterinary Inc, Princeton, NJ) daily for five postoperative days.

The animals in each study group were equally divided and killed at 4, 8, and 12 postoperative days. The dogs were killed in accordance with the laboratory animal guidelines. In control animals, the thoracic aorta was clamped distal to the left subclavian artery and proximal to the diaphragm. Intercostal artery branches from the thoracic aorta were then ligated and 20 mL of Microfil latex (Canton Biomedical, Boulder, CO) was injected into the thoracic aorta, filling the bronchial arteries and their mediastinal branches. In bronchial wrap animals, the pedicle arteries were examined for patency, cannulated with a 20F angiocatheter, flushed with heparinized saline solution, and then injected with 5 mL of Microfil. The heart and lungs were then removed from all animals en bloc while the integrity of the pedicle wraps was maintained. Transverse (A) and sagittal (B) diameters of the left main bronchus proximal to and at the anastomotic site were measured by a blinded investigator. The corresponding cross-sectional areas (CSAs) were calculated by the formula $A/2 \times B/2 \times \pi$. The degree of luminal reduction was determined by dividing the anastomotic CSA by the proximal CSA. The amount of submucosal latex present in the donor bronchus was graded either 0 (no latex), 1 (sparse or spotty latex), 2 (latex streaks), or 3 (confluent latex) (Fig 2). The latex was allowed to set for three hours, and the specimen was placed in formalin.

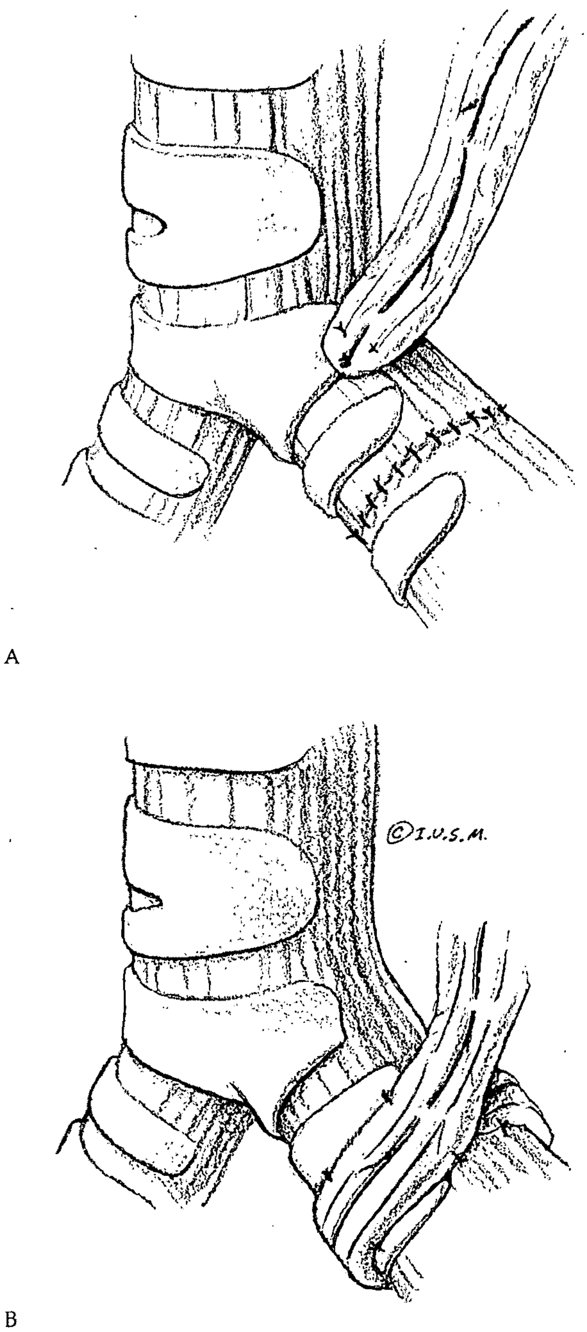

Fig 1. (A) Left main bronchial anastomosis with mobilized internal mammary artery pedicle graft after distal vessel ligation and pedicle division. (B) Orientation of internal mammary artery pedicle after circumferential wrapping of bronchial anastomosis.

Histological analysis was performed by a single pathologist who was blinded as to both the study group and

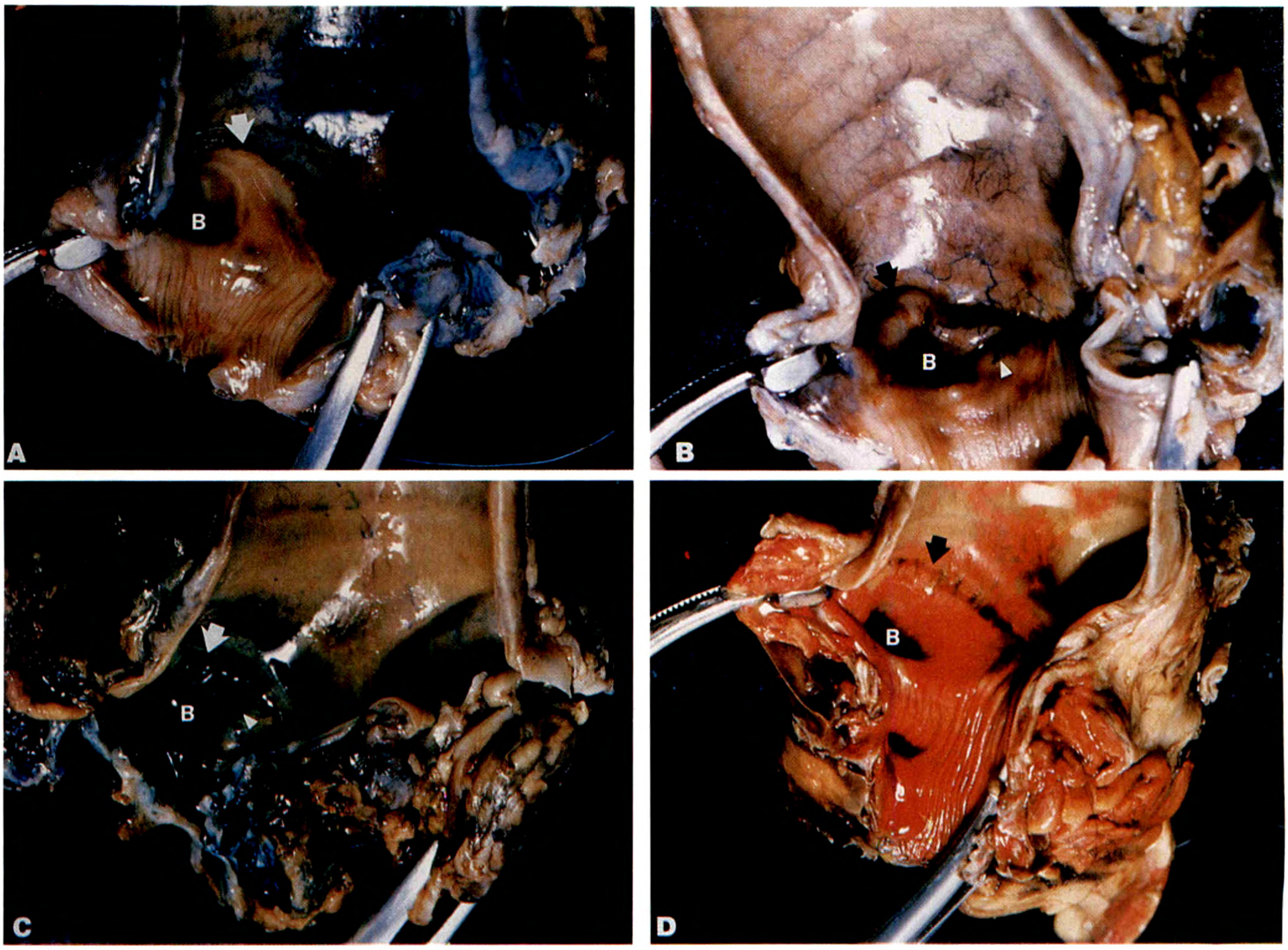

Fig 2. Submucosal vascular ingrowth grading system. (A) Grade 0—no latex (control, day 12). (B) Grade 1—sparse or spotty latex (control, day 8). (C) Grade 2—latex streaks (intercostal muscle, day 4), (D) Grade 3—confluent latex (omentum, day 12). (B = *bronchial lumen;* large arrow = *bronchial suture line;* small arrow = *submucosal vascular latex.)*

time interval of animal death. Hematoxylin and eosin-stained specimens were examined for the presence of Microfil within the submucosal vascular plexus and for mucosal integrity.

All statistical analyses were accomplished using the SAS software system. A Kruskal-Wallis test and Dunn's post hoc multiple comparison procedure were used to investigate the relationship between treatment group and revascularization category (grade 0–3). One-way analysis of variance and Duncan's multiple comparison procedure [12] were used to compare cross-sectional donor bronchial area between treatment groups, whereas simple linear regression was used to investigate the relationship between cross-sectional donor bronchial area and revascularization category (grade 0–3). Multivariate relationships between anastomotic cross-sectional area and the degree of submucosal revascularization, day of death, and treatment group were investigated using analysis of covariance.

Results

There was one premature death, which was unrelated to the operative procedure itself, and the animal was excluded from the study. All pedicle arteries remained patent. There was no bronchial dehiscence in any wrapped anastomosis; however, there was one frank anastomotic disruption (16.7%) in the control group.

Microfil injection into the collateral mediastinal arteries of control animals failed to reveal any capillary bridging across the anastomosis at postoperative day 4 (Fig 3). Sparse submucosal latex streaking (grade 0–1) was found in the ischemic bronchus of only 1 control animal killed on postoperative day 8. In contrast, at least 1 animal in every group with a wrapped anastomosis demonstrated grade 1 submucosal latex at 4 postoperative days. Moreover, 2

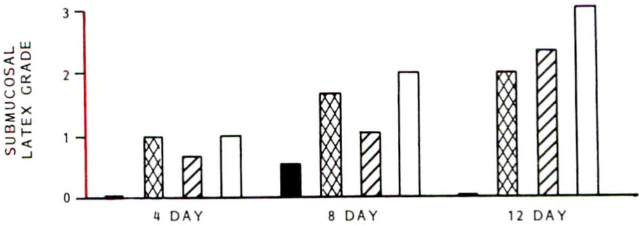

Fig 3. Mean submucosal vascular ingrowth by latex grading at time of death. (solid = *control,* crossed hatched = *intercostal muscle;* diagonal = *omentum;* open = *internal mammary artery.)*

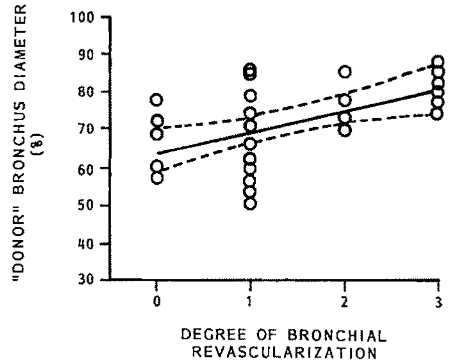

Fig 4. Scattergram plot of "donor" bronchus diameter with respect to the degree of submucosal vascular ingrowth including simple linear regression line with 95% confidence limits.

animals in every group with wrapped anastomoses had circumferential and confluent submucosal vascular ingrowth into the ischemic donor bronchus at or after postoperative day 8. Bronchi wrapped with the IMA pedicle appeared to have more consistent and progressive confluent vascular ingrowth than the other two pedicle grafts. Results obtained from the Kruskal-Wallis test indicated the presence of an overall significant difference in vascular ingrowth between the treatment groups ($p = 0.02$). Furthermore, Dunn's multiple comparison procedure demonstrated that there existed a trend for the vascular ingrowth observed using IMA, OM, and ICM pedicle grafts to be greater than that observed in the control group ($p = 0.09$).

The extent of submucosal vascular ingrowth influenced the degree of bronchial luminal narrowing at the anastomosis. All donor bronchi with grade 3 submucosal latex retained 81.2% ± 4.8% of original bronchial cross-sectional area as compared with bronchi with grade 2 (76.5% ± 6.6%), grade 1 (68.4% ± 10.9%), and grade 0 (65.0% ± 8.4%) (Fig 4). Simple linear regression demonstrated a significant trend of increasing bronchial anastomotic CSA with increasing grade of submucosal latex ($p = 0.002$). Application of analysis of covariance to these data, controlling for treatment group and day of death, indicated that in the presence of these other variables, the amount of submucosal latex remained significantly predictive of an increase in anastomotic CSA ($p = 0.04$).

Results obtained from one-way analysis of variance demonstrated a statistically significant difference ($p = 0.003$) between the observed CSA for IMA (84.5% ± 3.3%), ICM (66.9% ± 20.9%), OM (68.4% ± 8.3%), and control groups (70.2% ± 7.6%) (Fig 5). Furthermore, Duncan's multiple comparison procedure indicated that bronchi supported with IMA pedicle grafts had significantly greater CSA than those in the ICM, OM, and control groups, and that no significant differences existed between these latter three treatment groups. The treatment group remained a significant predictor ($p = 0.008$) of anastomotic stricture using analysis of covariance to control for day of death and amount of submucosal latex. Duncan's test again indicated that bronchi wrapped with IMA pedicle grafts had significantly greater CSA than bronchi in the other three treatment groups.

Histological evaluation of the bronchial mucosa in the control group demonstrated ulceration and necrosis in 3 animals, with only 3 dogs showing either regenerating epithelium or intermediate changes (50%). Random histological sections failed to detect submucosal latex in 5 of the 6 control animals. In contrast, bronchi supported by IMA external wraps demonstrated 5 animals with regenerating mucosa (83%), and only 1 with minimal focal ulceration. Similarly, 78% (7/9) of OM-wrapped and 89% (8/9) of ICM-wrapped bronchi had epithelial regeneration.

Comment

Bronchial healing after radical tracheobronchial operation is not infrequently complicated by anastomotic stenosis or even frank dehiscence. This has been attributed to ischemic injury secondary to interruption of the bronchial arterial circulation. Bronchial arteries originate from the aorta or intercostal arteries. They initially branch at the carina, then provide blood supply to main and segmental bronchi through a peribronchial adventitial plexus [1]. The distal main and first segmental bronchi, unlike the proximal main bronchi and carina, have bronchopulmonary arterial collaterals that provide an important source of bronchial blood supply if the bronchial arteries are divided. Without this dual blood supply, proximal main bronchial segments appear to be more susceptible to ischemic injury.

Both omental and intercostal artery pedicle wraps have previously been found to provide early capillary ingrowth into ischemic bronchial tissue and improve healing [5, 10, 11]. The current success of human lung transplantation has been attributed, to a large degree, to the technique of wrapping the bronchial anastomosis with these vascular tissue pedicles, thereby providing improved blood supply to the anastomosis and reducing the incidence of bronchial dehiscence. By necessity, omental wraps use celiotomy for mobilization with graft passage through a diaphragmatic defect resulting in a pleuroperitoneal communication. Additionally, cachectic, malnourished patients or patients with previous upper abdominal operation may not have sufficient omentum for bronchial coverage. Intercostal muscle pedicle grafts, based on an intercostal artery, have not previously demonstrated mu-

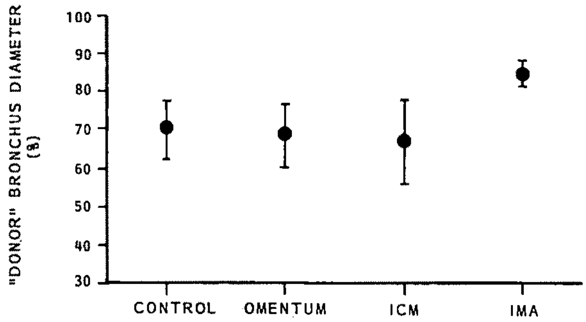

Fig 5. "Donor" bronchus diameter expressed as mean percentage of proximal bronchial diameter observed between treatment groups. (Bar = one standard deviation; ICM = intercostal muscle; IMA = internal mammary artery.)

cosal capillary ingrowth until the seventh postoperative day [10]. Moreover, this pedicle graft is based on a smaller, more tenuous vessel that may be easily damaged during mobilization or kinked when the pedicle is rotated into position around the bronchus. Although direct revascularization by bronchial arterial anastomosis has great theoretical appeal, this requires microvascular techniques without uniform reliability.

Diaphragmatic muscle wraps based on branches of the IMA have been described [13], but our purpose is to describe use of the IMA pedicle for bronchial revascularization. The IMA pedicle graft has several theoretical advantages. Its dissection and mobilization have become a routine part of most coronary artery bypass procedures, which may be done through either a sternotomy or posterior lateral thoracotomy incision. Because of its anatomical location and length, the internal mammary artery pedicle graft can circumferentially wrap any mediastinal tracheal or main bronchial suture line without tension, kinking, or distortion. In our experiment, the IMA pedicle graft demonstrated more consistent and progressive revascularization of the ischemic bronchus as compared with the other study treatments or a control (no wrap). This early revascularization resulted in improved preservation of bronchial mucosa, promoted normal healing, and minimized anastomotic stricture.

Confluent submucosal vascular ingrowth, provided by any of the three types of arterial based wraps, was found to be independently predictive of reducing bronchial anastomotic stenosis. Several animals with intercostal artery or omental wraps, however, had no capillary ingrowth that resulted in severe anastomotic strictures similar to controls. This provides further evidence that revascularization, even by indirect methods, promotes more normal bronchial healing.

Before assuming that these results will extrapolate clinically, one must be mindful of several important anatomical variations that exist between the human and canine IMA. First, the canine IMA pedicle has a well-developed muscle with a transparent pleural surface as opposed to that of humans, which is mainly fibrofatty tissue with a thick fascial layer lining the pleural surface that could potentially limit vascular ingrowth. Sparse muscle is found only on the inferior one third of its intrathoracic course. Additionally, the canine IMA diameter and flow is significantly greater per body weight than that of humans. Possibly more important are functional differences between the IMA pedicle graft and omental wraps with respect to reticuloendothelial function. Extensive phagocytic properties of the omentum are not present in the IMA pedicle graft, which theoretically may be important for local bacteriologic control, especially in immunosuppressed patients.

This study supports earlier evidence regarding the ability of omental and intercostal artery pedicle grafts to promote early indirect revascularization of ischemic bronchial tissue. Based on the findings of this study, the canine IMA pedicle graft appears to be an excellent intrathoracic source for early submucosal revascularization of the bronchus, providing a more consistent and progressive ingrowth pattern as compared with wraps currently used. It would seem prudent, however, to clinically evaluate IMA vascular pedicle ingrowth characteristics in nonimmunosuppressed patients undergoing radical tracheobronchial operation before the IMA's application in lung transplantation.

We thank Randy Bills and Stacy Wesemann for assistance in the operative phase of this study, as well as Myrtle Parsley for secretarial support.

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DISCUSSION

DR G. ALEXANDER PATTERSON (Toronto, Ont, Canada): I would like to compliment Dr Turrentine and associates for a very well-designed and executed study of a problem that I think is becoming increasingly important as the number of pulmonary transplant centers increases. In our own center, 3 of 27 single-lung and 8 of 17 double-lung recipients have had major airway complications. Three of these were fatal.

Oriani Lima, working with Dr Joel Cooper, has demonstrated the remarkable capacity of omentum to revascularize ischemic donor bronchi in a very short time. Dr Veith's group subsequently demonstrated reliable revascularization with intercostal muscle.

We have used omentopexy routinely, but in recent cases, we have employed a pedicle of anterior mediastinal fat and thymus on a superior-based pericardial flap. We continue to use omentum when this flap is of questionable substance or viability. The Pittsburgh group has employed IMA pedicles to encircle tracheal anastomoses after heart-lung and double-lung transplantation.

We are also learning about angiogenesis. Recent reports have described a lipid extract of omentum with remarkable angiogenic properties. We have recent experimental data suggesting that basic fibroblastic growth factor, a potent angiogenic agent, does not augment omental revascularization of transplanted tracheal segments. We have also found that improved pulmonary preservation appears to be associated with improved bronchial viability after lung allotransplantation. This is presumably due to increased preservation of microcirculation and collateral flow between the pulmonary artery and bronchial artery so well described by the Pittsburgh group.

I would like to make one further comment and ask a couple of questions. I hate to expose my statistical ignorance, but I would be a little cautious about assuming a superiority of the IMA pedicle in such a small number of animals killed at variable times.

I do not have any question that bronchial wrapping is important. I am not so certain that we can be categorical about what is the best flap at the moment.

I wonder if Dr Turrentine can tell me whether or not these animals were all heparinized and whether the lungs were flushed before extraction.

I am also rather in admiration of 31 left lung allografts done without any evidence of atrial thrombosis, and I wonder if you could comment on whether you had an incidence of atrial thrombosis in your transplanted animals.

You mentioned differences in cross-sectional area from group to group, but was there a difference in cross-sectional area from time to time? In other words, did cross-sectional area at four days differ from that observed at eight or 12 days, and if so, was there a difference between groups?

DR STANLEY C. FELL (Chappaqua, NY): I congratulate Turrentine and associates on demonstrating yet another method of revascularizing the bronchus. However, I do not believe their test was sufficiently rigorous. Only one of their control animals had a disruption. We have used the same experimental model that the Toronto group used, namely, the postpneumonectomy bronchial autograft, when we did a study on intercostal muscle flaps [Fell SC, Mollenkopf FP, Montefusco CM, et al. Revascularization of ischemic bronchial anastomoses by an intercostal pedicle flap. J Thorac Cardiovasc Surg 1985;90:72–8], and the control is absolutely lethal. So I would suggest that this study be repeated using the proper control model.

DR JOEL D. COOPER (St. Louis, MO): I was not sure whether steroids were used. Just to recap, in the original work that Dr Lima and co-workers in Toronto did with us, in the absence of steroids, canine reimplanted bronchi and transplanted bronchi did appear to heal well. It was the combination of postoperative steroids and ischemia that led to poor healing.

We evaluate stenosis at various times, but stenosis is a late manifestation of the ischemic injury. By 12 days it is not usually apparent. The stenosis occurred without steroids, even in the presence of good wound healing. When observed at 3 weeks or later, the cross-sectional area began to narrow, and in that group, the omental wrap prevented stenosis compared with no omental wrap. So a couple of different factors were mixed in.

Finally, science has crept up with clinical practice. We now know why the omentum, as Dr Patterson commented, works well. It is the angiogenesis factor, of which it has a very potent supply, though this is not unique to the omentum. Angiogenesis factor is known to be in all fatty tissues. One of the contributions of the late Lyman Brewer was the use of the pericardial fat pad, also rich in angiogenesis factor and blood supply. So I do not think the omentum is unique. It happens to be bulky and easy to use, but other tissues throughout the body may also be used as long as they have good blood supply.

Dr Turrentine, can you tell me about the effects of steroids? Also, did you keep some of the dogs longer than 12 days so that you might perhaps begin to see some of the stenotic complications that, we think, are late manifestations of ischemia?

DR TURRENTINE: I thank all the discussants for their comments. It is certainly an honor to have the pioneers of clinical lung transplantation discuss this work.

First, Dr Patterson, our technique was to heparinize, explant, and directly reimplant the lung soon after with ischemic times less than 45 minutes. We did experience problems with left atrial clot early on while developing this autotransplant model. After we changed the left atrial suture technique to a running horizontal mattress, which everts the muscular atrial wall, we found no incidence of left atrial thrombosis in any study animal.

Dr Cooper, we hesitated to use the term *bronchial stenosis*, and perhaps we should rather use the term *reduction in luminal area*. Histologically, ulcerating mucosa in these "stenotic" areas appeared heaped up, which we feel represented an ischemic insult rather than a true scarified stenosis.

We did not use steroids in this experimental protocol. Perhaps this would have made a more challenging model by placing the anastomosis at greater risk for dehiscence; however, the primary purpose of the study was to evaluate vascular ingrowth in the ischemic bronchial segment with a comparative analysis between the vascularized pedicles suitable for such use.

We have had 4 animals that have undergone IMA-wrapped bronchial anastomosis after left lung autotransplantation and were studied up to 3 months postoperatively. In these longer term experiments, we have observed that bronchial lumen diameters compare equally with those of 12-day animals whose bronchi were wrapped with similar grafts.

Thoracic Aortic Aneurysms After Acute Type A Aortic Dissection: Necessity for Follow-up

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Between April 1979 and May 1989, 86 patients underwent emergency operation for acute type A aortic dissection. Sixty-four (74.4%) survived. None of the survivors died of late aortic complications. Fifty-eight patients were followed 3 months to 10 years (mean follow-up, 3.2 years) postoperatively with computed tomography and digital subtraction angiography. Dilatation of the distal aorta (diameter size range, 6 to 10.5 cm) developed in 10 patients (17%). Six patients underwent replacement of the descending aorta 1 month to 21 months (mean period, 8.5 months) after aortic dissection repair. Two of them had third-stage thoracoabdominal replacement. In 2 patients, replacement of the descending aorta was scheduled; 1 died before reoperation and 1 refused the procedure. Two patients underwent aortic arch replacement; it is scheduled for another (fourth stage). There were no deaths among the patients having reoperation. The rate of indications for reoperation on the aorta downstream from the original repair 1 month to 6 years 4 months (median time, 9 months) after primary surgical intervention for acute type A aortic dissection was 24% (14 reoperations in 10 of 58 patients). This study underscores the importance of close follow-up of patients having operation for acute type A aortic dissection. Early recognition of progressive downstream aortic pathology permits effective prevention of aortic rupture and timely reoperation.

(Ann Thorac Surg 1990;49:580–4)

The aim of surgical intervention for acute type A aortic dissection is to prevent death by rupture of the ascending aorta. In the majority of patients, aortic dissection persists distal to the site of the primary operation. The survivors have a generalized disease, which often extends along the entire residual aorta and leads to further complications.

Early results of emergency operation for acute dissection have been improving over the years. Nevertheless, the late mortality related to aortic complications has remained strikingly high, accounting for up to one third of late deaths. Besides strict medical management to control risk factors such as arterial hypertension, a scrupulous long-term follow-up to detect changes in the residual aorta is mandatory. Only timely surgical reintervention can minimize the risk of late aortic complications.

Material and Methods

Between April 1979 and May 1989, 86 consecutive, unselected patients underwent emergency operation on the ascending aorta for acute type A aortic dissection. There were 69 men and 17 women with a mean age of 48.3 years (range, 20 to 73 years).

Based on the findings at operation, three basic procedures were performed. When the aortic root had a normal diameter, the valve was usually reconstructed and the ascending aorta was replaced or reconstructed by a technique described by us [1] and by Olinger and associates [2]. The aortic valve was replaced if it was morphologically deformed, known to be regurgitant before dissection, or destroyed beyond repair by dissection. In the case of ectasia of the aortic annulus, a composite graft bearing a mechanical bileaflet valve was inserted. If the upstream entry extended beyond the aortic clamp, the aortic arch was replaced proximally or totally.

The operative survivors were followed by computed tomography of the thoracic and abdominal aorta with contrast medium administered by the bolus technique, and by intravenous digital subtraction angiography of the entire aorta. In patients in whom the aortic valve had been reconstructed, transesophageal echocardiography was added to examine valve function.

These studies provided the standard before discharge for each patient. If no major pathological findings were present, the subsequent examinations were done 6 months postoperatively. For patients in whom the aorta was already dilated downstream, the interval was shortened to 3 months. Diameter and progression of aortic dilatation then determined the further intervals for control: 6 months or less for patients with evident aortic disease; 12 months for those with aortas in stable condition and diameters between 5 and 6 cm; and 24 months for those with aortas in stable condition and diameters smaller than 5 cm. No patient was denied follow-up irrespective of the degree of the residual pathological findings. More recently, nuclear magnetic resonance imaging was performed in some patients to reduce exposure

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Table 1. Follow-up Studies After Acute Type A Aortic Dissection

Study	Indication or Extent
Computed tomography	Thoracoabdominal aorta
Digital subtraction angiography	Entire aorta
Echocardiography (transesophageal)	Preserved aortic valve
Nuclear magnetic resonance	Select patients

to irradiation and to gain experience with this comparatively new diagnostic tool. The follow-up program after acute type A aortic dissection is summarized in Tables 1 and 2.

The indications for replacement of the affected aortic segment were based on three criteria: (1) aortic diameter exceeding 6 cm; (2) rapid progression of aortic diameter enlargement; and (3) symptomatic aortic aneurysm. Follow-up by the regimen just described was continued after reoperation to detect possible indications for third- or fourth-stage operations.

For the 6 patients who refused systematic follow-up, information concerning survival and state of health was obtained from the family doctor.

Results

During the 10-year period of this study, 86 patients underwent emergency operation for acute type A aortic dissection. In 23 (26.7%) of them, reconstruction of the ascending aorta with or without reconstruction of the aortic valve was performed. In 33 patients (38.4%), the ascending aorta was replaced by a tubular prosthetic graft, again with or without valve reconstruction. In 30 patients (34.9%), the aortic valve was replaced by a composite graft (n = 28) or by separate replacement of the valve and the ascending aorta (n = 2). The native aortic valve was retained in 56 (65.1%) of the 86 patients.

Sixteen patients (18.6%) required additional surgical intervention on the aortic arch with deep hypothermia and circulatory arrest. The procedures included distal graft anastomosis by the open technique (6 patients), proximal arch replacement (8 patients), and total arch replacement (2 patients) [3–6].

Overall operative mortality was 25.6% (22/86). There was, however, a dramatic learning effect over the 10 years. Of the 65 patients having operation since January 1, 1984, 13, or 20%, died; of the 29 patients operated on since January 1, 1987, 4, or 13.8%, died; and of 18 patients operated on since January 1, 1988, 2 died, an operative mortality of 11.1%. For operations on the aortic arch, overall operative mortality was 25% (4/16); there have been no deaths among the 7 patients undergoing operation since 1987.

Table 2. Schedule for Follow-up Studies

Time	Indication
Before discharge	Routine
Initial after discharge (mo)	
3	Dilated residual aorta
6	Normal aortic diameter
Subsequent (mo)	
Every 6	Progression of aortic disease
Every 12	Aortic diameter ≥5 cm
Every 24	Aortic diameter <5 cm

Table 3. Length of Follow-up for 58 Patients

Follow-up (yr)	No. of Patients
0.3–1	9
1–2	12
2–3	12
3–4	11
4–5	5
5–6	4
6–7	2
7–8	1
8–9	1
9–10	0
10–11	1

Late mortality was 3.1% (2/64). One 42-year-old patient on a regimen of Coumadin (crystalline warfarin sodium) died of intracranial hemorrhage 2 years after separate replacement of the aortic valve and ascending aorta. The other death occurred in a 42-year-old patient with Marfan's syndrome who had had composite graft replacement of the aortic valve and ascending aorta. Twenty-two months after this operation, follow-up revealed enlargement of the diameter of the descending aorta to 6 cm and mitral valve degeneration with marked regurgitation. The patient also had a severe ventricular arrhythmia not amenable to medical therapy. He was scheduled for replacement of both the mitral valve and the descending aorta, but died, presumably of the arrhythmia, at home.

Fifty-eight patients (58/64 survivors or 90.6%) were followed on a regular basis by computed tomography and digital subtraction angiography over a period ranging from 3 months to 10 years postoperatively. Six patients refused regular follow-up; all received sporadic follow-up examinations in other institutions. According to the family doctors, 5 of these patients were alive and in good health at the time of the study, and 1 had died of cerebral bleeding, as already mentioned.

The mean period of observation was 3.2 years. The distribution of follow-up years is shown in Table 3. Extensive dilatation of the aorta distal to the primary operative site developed in 10 (17%) of the 58 survivors followed. Aortic diameters ranged from 6 to 10.5 cm. The secondary procedures performed are shown in Table 4. Two male patients underwent total aortic arch replacement 5 and 8 months after primary operation for acute type A aortic dissection. Both had demonstrated severe hypertension and rapid progression of aortic diameter enlargement immediately distal to the primary repair.

Six patients underwent replacement of the descending aorta 1 month to 21 months (mean period, 8.5 months) after primary repair. Two of them subsequently had third-stage thoracoabdominal replacement: 1 at 4 years 9 months after acute dissection and 3 years 6 months after replacement of the descending aorta and the other, 4 months after the primary procedure and 1 month after replacement of the descending aorta. In 1 of the 6 patients, third-stage total arch replacement was performed 2 years 1 month after acute dissection and 2 years after replacement of the descending aorta. There were no deaths in these 11 reoperations.

Replacement of the descending aorta was scheduled for 2 patients. One died before reoperation 2 years after dissection. The other patient refuses the replacement procedure. His descending aorta had reached a diameter of greater than 6 cm 6 months after primary repair, and over the past 4 years, has increased to 7 cm.

One patient is scheduled for fourth-stage aortic arch replacement. He underwent reconstruction of the ascending aorta without the valve in November 1982. By January 1984, the descending aorta had reached a diameter of greater than 9 cm and was replaced. In August 1987, thoracoabdominal replacement was performed because of enlargement to more than 6 cm. The ascending aorta distal to the reconstructed part and the aortic arch show a slow but persistent increase in diameter, which has now reached 6.1 cm.

The mean interval to aneurysm formation was 18 months (range, 1 month to 6 years 4 months; median time, 9 months). The average interval from primary dissection to reintervention differed according to the aortic portion affected: descending aorta (n = 8), 9.9 months (range, 1 to 22 months); aortic arch (n = 4), 28.5 months (range, 5 to 76 months); and thoracoabdominal aorta (n = 2), 30.5 months (range, 4 to 57 months) (Table 5).

Two additional patients required reoperation on the ascending aorta because of failure of the primary repair. In 1 of them, one of the resuspended valve commissures tore out 6 weeks postoperatively, and composite graft replacement was performed. In the other, a woman with Marfan's syndrome, ectasia of the aortic annulus developed after reconstruction of the aortic valve and separate replacement of the ascending aorta. After composite graft replacement 5.5 years after primary repair, possibly septic leaks occurred at both coronary ostia and were repaired successfully. Thus, the total number of reoperations and scheduled reoperations after acute type A aortic dissection was 17 in 12 of 58 patients (17/58 or 29.3%; 12/58 or 20.7%): 14 for aneurysm formation in the thoracic aorta and 3 for complications at the primary site.

Table 4. Reoperations After Acute Type A Aortic Dissection in 58 Patients

Operation	No. of Patients
Performed	
Replacement of descending aorta	6
Replacement of thoracoabdominal aorta	2[a]
Replacement of aortic arch	3[b]
Scheduled	
Replacement of descending aorta	2
Died before performed (arrhythmia)	1
Refuses operation	1
Replacement of aortic arch (fourth stage)	1

[a] These 2 patients were among the 6 having replacement of the descending aorta. [b] One of these patients was among the 6 having replacement of the descending aorta.

Table 5. Intervals to Reoperation After Acute Type A Aortic Dissection

Segment Replaced	No. of Patients	Interval After Primary Operation (mo)	Average Interval[a] (mo)
Descending aorta			9.9
Performed (second stage)	6	1, 3, 3, 10, 13, 21	
Scheduled	2	6, 22	
Aortic arch			28.5
Performed	3		
Second stage	2	5, 8	
Third stage	1	25	
Scheduled (fourth stage)	1	76	
Thoracoabdominal aorta			
Performed (third stage)	2	4, 57	30.5

[a] The average interval from primary repair to reoperation was 18.4 months and the median interval, 9.0 months.

Comment

Acute type A aortic dissection remains one of the challenges of modern cardiovascular surgery. Emergency procedures for this condition are aimed primarily at the prevention of aortic rupture and therefore generally are limited to the area of highest risk, that is, the proximal portion of that vessel. After successful primary repair, the remainder of the aorta is left dissected in most instances, usually down into the iliac arteries [7].

Dilatation with ultimate rupture of the distal aorta is known to be the most common cause of late death after operation for acute dissection [7–10]. In the series of DeBakey and associates [8] comprising 527 patients operated on for dissection, rupture of the distal aneurysm accounted for the highest percentage of late deaths (29.3%). DeBakey and associates found different incidences of subsequent aneurysm formation depending on the type of dissection. For type I dissection, there was an incidence of 29.8% and for type II, 14%; the total incidence was 24.7%. The Stanford experience [9, 10] showed that replacement of the aorta in symptomatic or expanding dissection or both improved long-term survival. Sixteen percent of the patients underwent reoperation for a total of 27 procedures in 22 patients. Aortic rupture accounted for 18% of late deaths.

It is difficult to define the stage at which an aneurysm developing after dissection may rupture and therefore should be resected prophylactically. The critical diameters, however, can be derived from the experience gained with arteriosclerotic abdominal aneurysms. It has been shown that the risk of rupture within 1 year is 43% for aneurysms with diameters greater than 6 cm, 80% with diameters of 8 cm and greater, and 4% with diameters smaller than 5 cm [11]. This is also illustrated by the correlation demonstrated by Szilagyi and colleagues [12] between the risk of rupture and the size of the abdominal aortic aneurysm. In their series, 79.2% of ruptured aneurysms had a diameter greater than 6 cm.

McNamara and Pressler [13, 14], when studying the natural history of arteriosclerotic thoracic aortic aneurysms, documented a mean survival of less than 3 years without operation. Rupture of thoracic and abdominal aneurysms occurred with about the same frequency, but the prognosis for the former was found to be worse. Therefore, McNamara and Pressler favored early surgical intervention, especially if enlargement of the aneurysm was noted within 1 year. Parker [15] found that 85.7% of ruptured thoracic aneurysms in his series had diameters greater than 6 cm.

There is reason to believe that the prognosis for an aorta affected by dissection is even more unfavorable than that of arteriosclerotic vessels. In aortic dissection, the false channel is in the exterior half of the media. As a consequence, its outer wall is usually only about one fourth as thick as the original medial wall, a finding that by itself may explain the high incidence of rupture [14, 16]. Another specific feature of aortic dissection is the rapid acquisition of atheromatous changes in the wall of the false channel, exceeding within months the changes normally acquired within a lifetime [17]. Weakening of the aortic wall may be even more rapid in the presence of gross medial disease in Marfan's syndrome. In these patients, the necessity for close follow-up of the aorta has been stressed [18, 19].

As for the methods of follow-up, digital subtraction angiography is the obvious choice. It depicts the anatomy of the aorta, the relationship between true and false lumens, and possible anomalies of aortic branches [20]. On the other hand, computed tomography with administration of contrast medium by the bolus technique at the levels of interest is the best method for measuring size of aortic diameters [21, 22]. It also demonstrates thrombosis of the false lumen and the relationship of aneurysms to neighboring organs. The technique of nuclear magnetic resonance imaging is evolving rapidly and seems to integrate the advantages of the two techniques already mentioned [23–25]. However, its availability is still limited. With the technologies available today in cardiovascular surgery, the aorta can be entirely replaced with comparatively low risk [8, 26, 27]. New operative methods, such as the "elephant trunk technique," have been employed to facilitate some of our reoperations [28].

Our results demonstrate that patients who survive emergency operation for acute type A aortic dissection run a considerable risk of dying of late complications, especially aneurysm formation with aortic rupture. The descending aorta seems to be the area most prone to expand. Enlargement of the aortic diameter is to be expected within the first 2 years after the acute event. Long-term follow-up, however, shows that a slow progression in size also is possible, thus necessitating follow-up of patients for life.

Follow-up of these patients by cardiologists and practitioners often is unsystematic, and therefore it should be directed by the surgeon as the person who has to decide when to reintervene. This is borne out by our experience, in which timely reintervention has resulted in the lowest postoperative mortality from aortic rupture reported to date in the literature.

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DISCUSSION

DR GRAEME L. HAMMOND (New Haven, CT): The presentation reviews an extremely difficult and challenging surgical problem, that is, management of type A aortic dissections. Dr Heinemann and his colleagues describe an operative mortality of 26% for repair of, replacement of, or reconstructive procedures on the ascending aorta. These are commendable results. However, of the patients who died, how many died of intraoperative bleeding?

We have performed approximately 90 repairs of the ascending aorta, and during the past 3 years we have essentially eliminated our intraoperative bleeding problems by using the following techniques: First, we soak the proximal and distal ends of the aorta remaining after resection in 25% glutaraldehyde solution for seven minutes. This provides a very tough aorta to which to sew and prevents extension of suture holes in the aorta on release of the cross-clamp.

Second, when there is severe aortic insufficiency, we insert a composite valve-graft as does Dr Heinemann, except that we avoid the difficulties inherent in suturing the low-lying ostia of the coronary arteries to the Dacron graft by using the following technique: The saphenous vein is removed from the leg and placed in a bowl, and the ascending aorta is then resected. A No. 10 red-rubber catheter is threaded through the vein graft and inserted into the left coronary orifice, and the saphenous vein graft is sewn to the left coronary orifice using the red-rubber catheter as a stent. The stent is removed and the procedure is repeated with the right coronary orifice. The vein grafts are used to deliver cardioplegia during insertion of the conduit. Finally, the grafts are sewn to the anterior wall of the conduit.

We totally agree with the necessity for long-term follow-up of these patients. Dr Heinemann reports a 22% recurrence of aneurysm distal to the graft insertion site. I wonder if his patients are managed with long-term antihypertensive drugs and β-blockade postoperatively. We are very meticulous in this respect and find in our experience approximately a 10% recurrence of aneurysm distal to the graft in the aortic arch.

Finally, I point out that there are two ways distal dissection can present. One is by aneurysm formation, and the other is by obstruction of major vessels. In this regard, we have reviewed our experience with abdominal fenestration procedures to relieve vessel blockade. These data will be presented next month at the New England Surgical Society meeting in New Hampshire.

Dr Heinemann, have you had experience with fenestration procedures, which in our experience carry no mortality, for treating the sequelae of distal progression and vessel obstruction?

DR GILLES DREYFUS (Paris, France): We have found that about 10% of our patients treated for type A aortic dissection had a recurrent aortic dissection requiring treatment. There is a technical point I must stress; in France, we are using a biological glue that is not a fibrin glue. It is called GRF biological glue, a gelatin-resorcin form of glue, and it really reinforces the tissues. There are two advantages to its use: first, it diminishes the fragility of the tissues, and second, it helps seal the false lumen, which has been talked about previously.

I have a few questions for Dr Heinemann. Do you believe that only aneurysmal formations rupture? We think that even a normal aorta can rupture. What is your rate of paraplegia? Do you try to recognize the artery of Adamkiewicz before the operation for recurrent dissection? Last, did any patient die with such a follow-up but without reoperation?

DR HEINEMANN: Of our operative deaths, 6 patients died of intraoperative bleeding early in the series. This has not been a major problem during the last 2 to 3 years. Fibrin glue, with which we have considerable experience, has helped a lot.

Practically all our patients are on a regimen of antihypertensive drugs postoperatively, mainly β-blockers.

As for fenestration operations for obstructive lesions, we have done them twice during emergency repair of acute type A dissections. In an elective situation, we think that with our low mortality, if a patient is symptomatic as a result of pseudocoarctation of the distal aorta, we might just as well replace the affected aortic segment. This offers far better long-term results. In fact, the patients seen with obstructive lesions in dissection and their management are the subject of a study we have prepared.

We have found the elephant trunk technique very helpful for replacing the downstream aorta in a step-by-step fashion. There were no bleeding problems with that technique either.

In summary, close follow-up reveals that aortic aneurysms develop in a considerable proportion of patients after acute type A aortic dissection. Our aggressive surgical approach has resulted in the lowest late mortality reported to date.

Effects of Intraoperative Plasmapheresis on Blood Loss in Cardiac Surgery

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Intraoperative platelet-rich plasmapheresis allows autotransfusion of fresh, undamaged platelets and clotting factors at the completion of the operation. To evaluate this technology, we randomly assigned 100 consecutive patients who were to undergo an elective coronary bypass procedure and had normal clotting studies into the experimental (plasmapheresis) or the control group. Characteristics of both groups were similar, including average age (61.4 years versus 61.3 years [experimental versus control group]), sex (78% male versus 74% male), preoperative weight (80.9 kg versus 80.2 kg), preoperative red cell mass (1,989 mL versus 1,890 mL), perfusion time (102 minutes versus 106 minutes), and coagulation studies. Both internal mammary arteries were used in 68% of the patients. All patients had preoperative and postoperative blood volume determinations and complete clotting studies. Sixty-two variables related to bleeding were analyzed. Strict indications for transfusion were a hemoglobin level less than 7 g/100 mL in patients younger than 70 years and a hemoglobin level less than 8 g/100 mL in patients older than 70 years. The group receiving intraoperative plasmapheresis had a significant reduction in operative red cell mass loss (1,050 ± 43 mL versus 1,226 ± 61 mL; $p = 0.021$), a reduction in the average homologous transfusion (0.67 ± 0.15 unit versus 1.8 ± 0.25 units; $p = 0.0002$), and an increase in the percentage of patients not requiring blood transfusions (66% versus 32%; $p = 0.001$). This technique is useful in reducing postoperative blood loss and homologous transfusions.

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Recent awareness of transfusion-related diseases has made the reduction or elimination of homologous blood transfusions (HBTs) extremely desirable. Although essential to the reduction of operative blood loss, precise surgical technique alone is insufficient to prevent the need for HBTs. Application of blood conservation methods markedly reduces the need for homologous transfusions [1–6]. These techniques, designed to limit HBTs, focus primarily on retrieving and reinfusing blood lost during and immediately after operation [7] and on allowing normovolemic anemia [3, 6]. Most HBTs are required after the patient is no longer in the operating room because hemoglobin concentration steadily decreases from blood losses after chest closure [5, 8]. Thus, methods that reduce postoperative blood loss should be useful supplements to current blood conservation techniques.

Postoperative bleeding after cardiopulmonary bypass commonly results from damaged platelets [9–11]. Several nonrandomized reports have shown that intraoperative plasmapheresis with autotransfusion of platelet-rich plasma raises the postoperative platelet count [12–14] and reduces the need for HBTs [12, 14–16]. This randomized prospective study was designed to determine the quantitative effects of intraoperative plasmapheresis with reinfusion of autologous platelet-rich plasma on operative blood loss and HBTs for cardiac surgical patients.

Material and Methods

Between January and June 1987, 100 consecutive patients having elective coronary artery bypass operations were randomly assigned to an experimental (plasmapheresis) or control group. Patients with abnormal clotting studies were excluded. Conventional blood conservation methods, including hemodilution, intraoperative blood salvage, and reinfusion of shed mediastinal blood for the first four hours postoperatively, were used for all patients.

Plasmapheresis was started in the experimental group after induction of anesthesia and was terminated either at the accumulation of 1,000 mL of platelet-rich plasma or at the administration of heparin sodium. A plasma separator (Haemonetics model 5000) was used to collect platelet-rich plasma. Most patients required volume replacement with 5% albumin during plasmapheresis to maintain hemodynamic stability. All study patients tolerated this procedure. The platelet-rich plasma, stored at room temperature, was reinfused when the sternum was closed. Platelet counts and total protein values were determined from the platelet-rich plasma.

Two methods were used to determine red blood cell (RBC) mass values. Preoperative RBC mass values were calculated using the radioiodinated serum albumin method, and postoperative RBC mass values were determined by the radiochromium (^{51}Cr) method. Preoperative and postoperative RBC values were determined by both methods in the first 20 patients. The preoperative RBC volumes as ascertained by the two methods showed

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agreement, but immediate postoperative values averaged higher with the radioiodinated serum albumin method. Therefore, to reduce costs, preoperative testing was done by the less expensive radioiodinated serum albumin method only. To prevent inaccuracies in ^{51}Cr RBC mass determinations as a result of red cell damage during the pump run, blood samples were obtained preoperatively and were stored at 4°C in the blood bank for later labeling and reinjection. The postoperative RBC values were determined shortly after the patient's arrival in the intensive care unit. Each unit of homologous RBCs was counted as 260 mL of RBCs. All patients had hemograms and complete clotting studies performed preoperatively and postoperatively. The volumes of chest tube drainage were monitored at 4, 18, and 24 hours. Hematocrit determination was performed on the chest tube drainage at 18 hours postoperatively. Strict indications for HBTs were a hemoglobin level less than 7 g/100 mL in patients younger than 70 years and a hemoglobin level less than 8 g/100 mL in patients older than 70 years. Hemoglobin level was maintained at 10 g/100 mL in 2 patients with respiratory failure (1 patient in each group). Homologous platelet transfusions were administered if the platelet count was less than 100 × 10^9/L in a patient with active bleeding.

Operative techniques were identical in the two groups. Membrane oxygenators were used in all operations. Blood cardioplegia with added potassium was supplemented by topical cold saline solution. Hemodilution during extracorporeal circulation was allowed to reach a hematocrit of 0.21 before transfusion.

Student's t test was the primary statistical tool used in this research. Significance levels were determined using equal variances unless the hypothesis of equal variances was rejected at the 0.05 level. Multiple regression analyses and χ^2 tests were used as appropriate.

Results

Analyses of 62 variables related to bleeding showed that the composition of the experimental and control groups was similar. Table 1 shows the results of analysis by t test of some important independent variables. Preoperative RBC mass, age, sex, and weight, which have been shown to be important in transfusion requirements [8], were not significantly different between the two groups. Because elderly patients received transfusions at higher hemoglobin values, it is important to note that more septuagenarians were in the experimental group (24% versus 18%; p = 0.62). More patients in the plasmapheresis group took aspirin within 48 hours of operation than in the control group (34% versus 12%; p = 0.017). There were no noticeable differences, however, in operative blood loss or the need for HBTs between those who took aspirin and those who did not.

The average time to complete plasmapheresis was 57 ± 15 minutes (range, 30 to 92 minutes). There was sufficient time for plasmapheresis during preparation of both internal mammary artery grafts. In the majority of patients undergoing bilateral internal mammary artery grafting, plasmapheresis was done through the side port of a Swan-Ganz introducer. For patients for whom there was less preparation time available before bypass, plasmapheresis was performed by insertion of an additional catheter.

Table 1. Preoperative Characteristics of the Two Study Groups[a]

Variable	Control	Experimental	t Value	p Value
Preop RBC mass (mL)	1,890 ± 65	1,989 ± 61	−1.110	0.27
Age (yr)	61.27 ± 1.5	61.37 ± 1.3	−0.055	0.96
Sex (male = 1)	0.735 ± 0.06	0.784 ± 0.06	−0.576	0.57
Preop hemoglobin (g/100 mL)	13.9 ± 0.19	13.8 ± 0.22	0.748	0.46
Preop hematocrit	0.412 ± 0.055	0.402 ± 0.059	1.165	0.25
Clotting time (min)	7.3 ± 0.49	7.0 ± 0.55	0.319	0.75
Bleeding time (min)	5.9 ± 0.36	5.7 ± 0.22	0.563	0.58
Preop platelet count (×10^9/L)	253 ± 12	258 ± 80	−0.296	0.77
Preop weight (kg)	80.2 ± 2.5	80.9 ± 1.7	0.221	0.83
Pump time (min)	106 ± 5.7	102 ± 4.7	0.488	0.63

[a] Data are shown as the mean ± the standard error of the mean.

RBC = red blood cell.

The plasmapheresis group had less operative blood loss than the control group (1,050 ± 43 mL versus 1,226 ± 61 mL; p = 0.02) (Table 2). The operative RBC loss was determined by subtracting the postoperative RBC mass values from the preoperative RBC mass values plus the intraoperative homologous transfused RBC mass. Similar postoperative RBC mass values (985 ± 38 mL versus 992 ± 35 mL; p = 0.90) suggested that the indications for HBT were followed and that the control group was not over-

Table 2. Results in the Two Study Groups[a]

Variable	Control	Experimental	t Value	p Value
Op RBC loss (mL)	1,226 ± 61	1,050 ± 43	2.362	0.021
RBC HBTs (units)	1.8 ± 0.25	0.67 ± 0.15	3.845	0.0002
% Patients given HBTs	0.35 ± 0.07	0.67 ± 0.07	3.35	0.001
Clotting factors (units)	0.35 ± 0.15	0.04 ±0.04	2.018	0.049
% Patients given homologous clotting factor transfusions	0.12 ± 0.05	0.02 ± 0.02	2.008	0.049
Intraop autologous transfusion volume (mL)	652 ± 43	583 ± 36	1.235	0.220

[a] Data are shown as the mean ± the standard error of the mean.

HBTs = homologous blood transfusions; RBC = red blood cell.

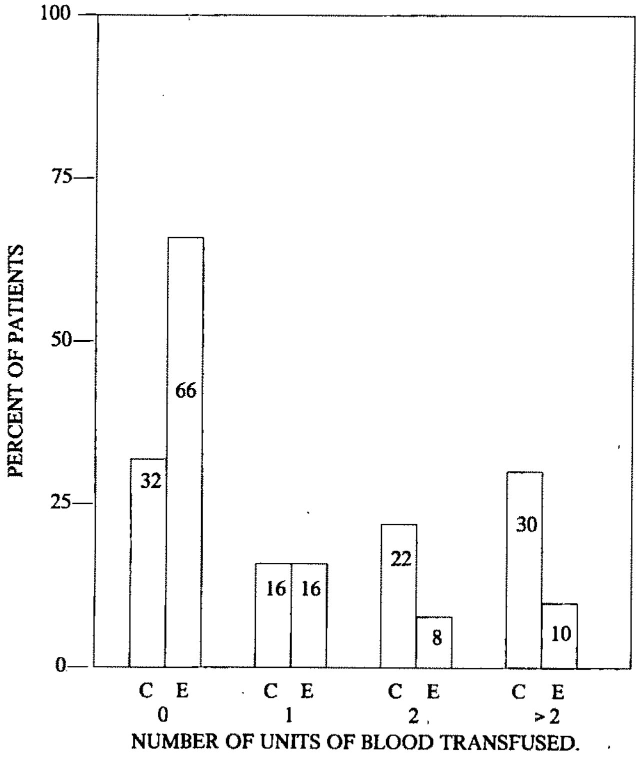

Fig 1. Distribution of patients by study group and number of units of homologous blood transfused. (C = control; E = experimental.)

transfused. Although the control group had a greater volume of intraoperative blood salvage, the difference was not significant. The estimate by the operating room nurses of operative blood losses by the gravimetric technique was slightly higher in the experimental group (598 ± 36 mL versus 480 ± 35 mL; $p = 0.21$). These values are estimates and are frequently inaccurate, but are included for completeness.

Most importantly, plasmapheresis patients received fewer HBTs than the control patients (0.67 ± 0.15 unit versus 1.80 ± 0.25 units; $p = 0.0002$). This reduction allowed approximately twice the number of patients to be discharged without exposure to banked blood (66% versus 32%; $p = 0.001$). Moreover, as shown in Figure 1, the majority of the plasmapheresis patients given transfusions received fewer units of blood than did the control patients. Eighty-one percent of all study patients had no HBTs in the operating room. Only 21% of the total units of homologous RBCs were administered in the operating room. When arranged according to preoperative RBC mass (Table 3), the data show a decreased percentage of patients requiring HBTs in all subsets with an RBC volume less than 2,500 mL in the plasmapheresis group.

Postoperative hemoglobin values were approximately the same in both the immediate postoperative period (9.8 g/100 mL versus 9.7 g/100 mL) and just before discharge (9.7 g/100 mL versus 9.4 g/100 mL) (Table 4). The 24-hour mediastinal drainage volumes were smaller and the drainage hematocrits lower in the plasmapheresis patients. These decreased mediastinal losses and the decreased losses in the operating room after administration of plasmapheresate most likely explain the need for fewer HBTs in the plasmapheresis group.

Table 3. Distribution of Patients and Percentages Requiring Homologous Blood Transfusion by Group and Preoperative Red Cell Volume[a]

Preop RBC Volume (mL)	Control	Experimental
<1,500	11 (82)	8 (63)
1,500–1,999	20 (80)	20 (45)
2,000–2,499	15 (40)	18 (22)
>2,500	4 (0)	4 (0)

[a] Numbers in parentheses are percentages of patients given homologous blood transfusions.

RBC = red blood cell.

Postoperative clotting studies, including activated clotting, prothrombin, and partial thromboplastin times, were similar in the two groups. Although not significant, postoperative platelet counts were higher in the experimental group (see Table 4). Six patients in the control group and 1 patient in the experimental group received homologous platelet transfusions. Three patients required reexploration for excessive mediastinal bleeding, 2 in the control group and 1 in the plasmapheresis group. The patient in the plasmapheresis group had bleeding from a branch of an internal mammary artery pedicle.

There were no hospital deaths. After discharge from the hospital, 1 patient in the plasmapheresis group died of a massive pulmonary embolus; the cause of death was substantiated at postmortem examination. Factors influencing RBC volume lost that were shown to be important by univariate analyses were examined by multiple regression analysis. The preoperative RBC volume ($p = 0.0001$), study group ($p = 0.0011$), and age ($p = 0.0136$) were significant.

Table 4. Univariate Analysis of Postoperative Values[a]

Variable	Control	Experimental	t Value	p Value
Hemoglobin level (g/100 mL)				
Immediate postop	9.8 ± 0.23	9.7 ± 0.22	0.292	0.77
Discharge	9.7 ± 0.17	9.4 ± 0.19	1.1863	0.24
Platelet count immediately postop ($\times 10^9$/L)	152 ± 7.6	165 ± 6.5	−1.3772	0.17
Chest tube hematocrit[b]	0.114 ± 0.017	0.068 ± 0.013	2.1295	0.04
Chest tube volume (mL)[c]	929 ± 96	828 ± 80	0.807	0.42

[a] Data are shown as the mean ± the standard error of the mean. [b] This is the hematocrit of the mediastinal drainage 18 hours postoperatively. [c] This is the chest tube drainage 24 hours postoperatively.

Comment

All methods used to reduce or possibly eliminate HBTs fall into three categories: increasing preoperative RBC mass, allowing postoperative anemia, or decreasing total perioperative RBC losses. First, the preoperative RBC mass can be increased by correction of anemias and use of banked autologous donations. All interventions to increase RBC mass before operation require time that frequently is not available for cardiac surgical patients [15, 17]. Moreover, a comprehensive survey of autologous practices by blood banks revealed that 49% of facilities exclude donors with a history of angina pectoris [18].

Second, allowing normovolemic anemia during the hospital course reduces the indications for HBTs. Although this approach is very useful, the limitation for allowing anemia is 7 g/100 mL; below this, detrimental effects can occur [19].

The major focus of blood conservation methods, however, falls into the category of reducing the volume of RBCs lost until hematopoiesis on postoperative day 4 or 5 begins reversing the anemia [20]. Precise surgical technique, hemodilution, and return of all pump perfusate reduce intraoperative losses. Postoperative blood losses can be minimized by reinfusing mediastinal drainage, accurately reversing anticoagulation, and reducing the number of laboratory tests. All of these measures were used in the present study.

Other investigators [5, 8] using these techniques found that few HBTs were required in the operating room. In this study, the majority of HBTs were necessary as a result of decreasing hemoglobin levels after sternal closure. Therefore, stabilizing postoperative RBC mass by limiting mediastinal blood loss should reduce the need for HBTs. The amount of mediastinal bleeding correlates with the number of functional platelets remaining after operation [9–11]. Cardiopulmonary bypass compounds the problem by damaging the clotting mechanism, which further increases mediastinal drainage. Correcting clotting deficiencies by homologous platelet transfusion is an undesirable solution. Besides being expensive, infusions of random donor platelets transmit disease as frequently as RBC transfusions on a per unit basis. Cardiac surgical patients given clotting factor and platelet transfusions have significantly higher rates of postoperative hepatitis [21].

Theoretically, sequestration of a substantial portion of a patient's own clotting mechanism from pump damage to reduce postoperative bleeding is an appealing concept. Studies [1, 2, 4, 5, 22] using intraoperative phlebotomy with reinfusion of fresh whole blood after bypass have shown reductions in HBTs. In some reports, patients given fresh autologous blood had increased postoperative platelet counts [22–24] and decreased mediastinal drainage [4, 22–24]. Platelet function after cardiopulmonary bypass improved more from infusion of 1 unit of fresh autologous blood than from transfusion of 5 units of homologous platelets [25]. Other studies [26, 27] using intraoperative phlebotomy have shown no benefit if heparin, which causes platelet dysfunction, was used as the anticoagulant. Perhaps because the majority of other routine clotting tests return to normal postoperatively, no improvement has been shown in prothrombin and partial thromboplastin times with intraoperative phlebotomy. Assuming benefits are dose related, the volume and the quality of autologous clotting elements become important. The volume of whole blood that can be removed before cardiopulmonary bypass is limited. The maximum volume removed in the previous studies was 1,000 mL or approximately 20% of an adult patient's total blood volume.

Developed in 1968, the plateletpheresis technique [28] allows selective collection of large quantities of platelets from a single donor. Single-donor homologous platelet infusions in cardiac surgical patients elevated the postoperative platelet counts and reduced postoperative bleeding [13]. This reduces but does not eliminate patient risk, as contrasted to autologous donations. In 1977, Harke and associates [12] used intraoperative autologous plateletpheresis in 17 patients. They found that the postoperative platelet counts were higher, blood loss was reduced by 50%, and the intraoperative screen-filtration pressures were lower than in the control patients. Others [29] verified that with this technique, the platelet counts dropped less postoperatively, and levels of clotting factors VII and XII were higher.

Cohn and associates [30] infused relatively small quantities (average, 337 mL) of autologous platelet-rich plasma and found the need for HBTs did not decrease significantly (4.1 units versus 5.2 units). They suggested that additional studies be done using larger-volume transfusions of platelet-rich plasma to evaluate the technique. More recently, Giordano and associates [15] used 500 mL of platelet-rich plasma in a study of a much larger group of patients. They reported a 50% reduction in HBTs. Furthermore, they [16] found the use of autologous platelet-rich plasma augmented other blood conservation techniques. However, these past reports are not randomized and rely heavily on the comparisons of homologous blood products used. Such comparisons are important but may depend on sometimes inconsistent indications for transfusion.

Many variables, including age, sex, weight, and preoperative hemoglobin level, influence the need for HBTs [8]. The most important determinant is the preoperative RBC volume [8]. In the present study, these variables were distributed equally in the control and experimental groups. The exact indications for HBTs were strictly followed, thereby limiting biases that could influence the decision to transfuse. Similar postoperative RBC mass values in both groups indicated that the control group was not overtransfused. The calculated volume of RBCs lost is approximately the difference seen in the amount of homologous blood transfused.

The platelet-rich plasma samples averaged $2.5 \pm 0.35 \times 10^{11}$ platelets, which is the approximate equivalent of 5 homologous platelet units. This quantity is approximately 30% of the average patient's total circulating platelets and should be sufficient to constitute a therapeutic quantity.

Concurrent with pressures to reduce HBTs, changes in the patient population needing cardiac operations hinder achievement of that goal. Cardiac operations are being performed on more elderly patients, on more female patients, and on more patients requiring difficult procedures. Possibly, the solution may be to apply the newer blood conservation methods more stringently. In this study, intraoperative plasmapheresis provided modest but significant reductions in blood loss and in the need for HBTs. This technique supplemented current blood conservation measures and significantly reduced HBTs in a program in which blood conservation is actively practiced. The most noticeable effect was the greater percentage of patients who were discharged without needing any homologous blood products.

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DISCUSSION

DR ROBERT L. THURER (Boston, MA): This study suggests that the sequestration and return of autologous platelet-rich plasma reduces bleeding and decreases the need for RBC transfusions. Dr Jones and his group are to be congratulated for investigating patients having modern operations using several blood conservation methods. Despite its apparent success, however, the study troubles me.

First, we and others have shown that more than 90% of coronary patients can have operations using few or no RBCs without platelet transfusion. Platelet use is related more to local preference than demonstrated need or benefit. Goodnough recently studied transfusion practice in first-time coronary bypass patients at eight institutions. Eighteen percent of patients received platelets, but institutional variation ranged from 0% to 80%. Bypass-induced platelet dysfunction does not require treatment in most cases. This study should not be interpreted as supportive of routine platelet transfusion to these patients.

Second, we are told that most transfusions are required after operation because hemoglobin concentration steadily decreases from blood losses after chest closure. Data from this study and other studies show that most blood loss occurs during operation. I believe that the postoperative fall in hemoglobin level is more often due to vasodilatation and equilibration than bleeding. In fact, despite the intervention, postoperative chest drainage did not differ between the groups. The hematocrit of the drainage was lower in the experimental group but, at the time measured, the difference was too low to be significant. The intervention did not reduce postoperative blood loss.

Last, the RBC mass determinations showed that control patients had more intraoperative blood loss. However, the sequestered plasma was not returned until sternal closure and therefore could not have reduced intraoperative bleeding.

We are left with the possibility that either by chance or due to the unblinded nature of the study, the groups were not equivalent and the control group merely bled more and was transfused more, with the intervention having no effect, or that blood loss was reduced by the removal of the platelet-rich plasma rather than by its return.

Although seemingly incongruous, possible explanations are that the experimental group had lower hematocrits during and after bypass and therefore lost less RBC mass, or that the experimental group was more completely anticoagulated because of removal of 30% of their clotting factors and had better protection of the circulating factors remaining.

Though the initial results are appealing, I do not believe this study supports the use of the technique. The place this method will find in the armamentarium of blood conservation, perhaps in more complex procedures, deserves further investigation.

DR JONES: I wish to allay Dr Thurer's fears—this paper does not recommend the routine use of homologous platelets. In fact, the purpose of the technique is to reduce the need for all homologous blood products. To clarify further, intraoperative RBC losses included drainage for the first few hours after sternal closure.

I am surprised that Dr Thurer concluded that our control group was overtransfused, particularly when he recently described that he transfuses 82% of coronary bypass patients and uses an average of 3 units of homologous blood per patient. Also, we made no attempt to quantitate and compare total blood losses with the volumes transfused. Per patient, the amount of transfused blood differed by merely 312 mL between the control and experimental groups.

True, a blinded study would be ideal but impossible to design under the circumstances. However, all evidence suggests that the HBT indications were strictly followed.

I believe that others will find this technique useful. In our study, most patients exhibited postoperative RBC mass values that bordered the minimal amount necessary before transfusion. Therefore, techniques that allow saving only 200 to 300 mL of RBCs can determine whether a patient will require HBTs.

Can Computed Tomography of the Chest Stage Lung Cancer?—Yes and No

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To determine the accuracy of computed tomography (CT) of the chest in the staging of lung cancer, we studied 418 patients with primary pulmonary carcinoma between 1979 and 1986. Each had a preoperative scan performed before detailed operative staging. Each CT scan was analyzed for components of the current TNM staging system. Computed tomography sensitivity and specificity for mediastinal lymph node metastasis were 84.4% and 84.1%, with corresponding positive and negative predictive accuracies of 68.7% and 92.9%, respectively. When TNM stages were derived from CT scans, only 190 of 418 (45.4%) completely agreed with operative staging. An additional 53 of 418 (12.7%) predicted the correct stage, although components of the TNM system were incorrect. In 94 of 418 scans (22.5%) CT overestimated the stage, whereas in 81 (19.4%) CT downgraded the stage. Computed tomography suggested metastatic lesions in liver, lung, adrenal gland, bone, or abdominal lymph nodes in 40 of 373 scans (10.7%); only five of 40 (12.5%) had documented metastasis. In summary, CT of the chest cannot accurately stage primary lung carcinoma according to the TNM classification. Because the negative predictive accuracy for mediastinal lymph node metastasis remains high (92.9%), invasive staging can be deferred for definitive thoracotomy when no lymphadenopathy is evident on CT. The high negative predictive accuracy for scans of the chest and upper abdomen makes CT a useful tool for exclusion of metastatic disease.

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Although computed tomography (CT) of the chest has been available since 1974, its definitive role in the staging of lung carcinoma remains controversial. While some reports in the literature praise its usefulness, others decry its inadequacies [1–15]. As a radiologic tool, CT is often expected to yield information about the possibility of malignant mediastinal and hilar adenopathy, invasion of thoracic structures by the primary tumor, or potential metastatic disease. To understand its usefulness in planning surgical approaches to primary lung carcinoma, we reviewed the records of 418 patients with detailed operative and CT staging.

Material and Methods

Between November 1979 and December 1986, 418 patients had detailed preoperative CT scans followed by a diagnostic or therapeutic operation for primary lung cancer. The mean patient age was 61.8 ± 10.2 years (range, 17 to 85 years). The ratio of male to female patients was 2.0:1, and the ratio of whites to blacks was 2.4:1. Cell types included adenocarcinoma (175 of 418, 41.9%), squamous carcinoma (137, 32.8%), undifferentiated (26, 6.2%), bronchoalveolar (24, 5.7%), small cell (18, 4.3%), large cell (15, 3.6%), carcinoid (8, 1.9%), and mixed-cell tumors (15, 3.6%). A standard posterolateral thoracotomy was performed in 364 of 418 (87.1%) patients (210 right, 57.7%; 154 left, 42.3%). Cervical mediastinoscopy or anterior mediastinotomy was performed alone in 54 patients (12.9%) and combined with a thoracotomy in 22 (5.3%). Extensive lymph node resections were performed during posterolateral thoracotomies in a manner similar to that described by Martini and McCormack [16]. Subcarinal and bilateral paratracheal lymph nodes were biopsied during mediastinoscopy; aortopulmonary and anterior aortic nodes were sampled at anterior mediastinoscopy. For uniformity, all CT reports and operative stages performed earlier in this series were converted to the New International Staging System for Lung Cancer [17]. To further ensure the accuracy of staging, a minimum 2-year follow-up was allowed for each surviving patient.

Computed tomographic examinations were performed on three different machines. From late 1979 to October 1982, all studies were performed on an EMI 5005, a second-generation scanner, which was replaced in 1982 by a fourth-generation Picker 1200-SX. Additional General Electric 9800 third-generation scanners were added in March 1985. The number of CT scans performed during individual years varied as follows: 1979, 29 of 418 (6.9%); 1980, 38 (9.1%); 1981, 22 (5.3%); 1982, 46 (11.0%); 1983, 64 (15.3%); 1984, 64 (15.3%); 1985, 84 (20.1%); and 1986, 71 (17.0%). Techniques for scanning evolved from 1979 to the present. The protocol in use by the end of the study was as follows. All patients without iodine allergies re-

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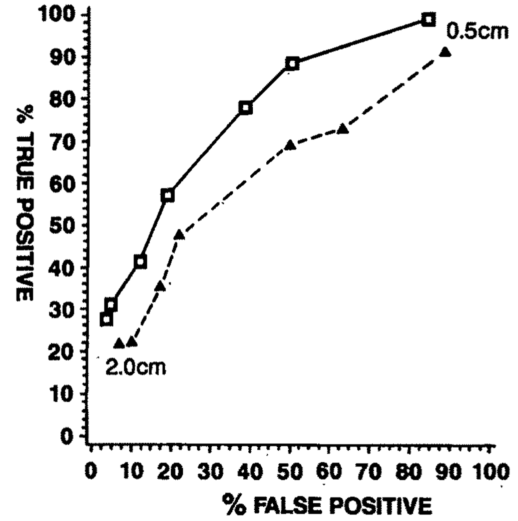


Fig 1. Receiver operating characteristic curves for the short-axis dimension of mediastinal (▲—▲) and hilar lymph nodes (□—□). The uppermost points on both curves reflect lymph nodes of 0.5-cm short-axis diameter. Curves descend in 0.25-cm increments to 2.0-cm diameter lymph nodes at the bottom of each curve.

ceived 150 mL of 60% iodinated contrast medium intravenously 20 seconds before the onset of scanning. If automated injectors were used, the injection rate was 2 mL/s. Scanning began at the level of the lower pole of the right hilum and extended cephalad to the thoracic inlet. It was continued from the lower pole of the right hilum downward to include both adrenal glands in most instances. Scanned sections were 1 cm thick. The scan times were 1.2 seconds for the Picker device and 2.0 seconds for the General Electric devices. Both had a 6-second interscan delay. Scanning was performed in suspended inspiration. Mediastinal and hilar lymph nodes 1.0 cm or less in short-axis diameter were considered normal. Current chest roentgenograms and conventional tomograms when performed (n = 169) also were examined for evidence of hilar and mediastinal lymph node enlargement. The reliability determinations for sensitivity, specificity, positive and negative predictive values, and accuracy described elsewhere were calculated for all CT scans, chest roentgenograms, and conventional tomograms [9]. Receiver operating characteristic curves [18] were constructed for short-axis dimensions of mediastinal and hilar lymph nodes in 0.25-cm increments from 0.5 to 2.0 cm (Fig 1).

Table 1. Reliability Estimates for Various Imaging Techniques in Detection of Malignant Adenopathy in Lung Carcinoma

Adenopathy (Technique)	Sensitivity (%)	Specificity (%)	PPA (%)	NPA (%)	Accuracy (%)
Mediastinal lymphadenopathy					
Computed tomography	84.4	84.1	68.7	92.9	84.2
Conventional tomography	51.2	93.6	73.3	84.9	82.8
Plain chest roentgenogram	40.2	99.0	94.6	80.1	81.9
Hilar lymphadenopathy					
Computed tomography	52.2	91.7	68.1	85.0	81.7
Conventional tomography	41.0	81.6	43.2	80.2	71.2
Plain chest roentgenogram	36.4	94.2	68.6	81.0	79.3

NPA = negative predictive accuracy; PPA = positive predictive accuracy.

Table 2. Computed Tomography Reliability Estimates for Detection of Malignant Mediastinal Adenopathy in Various Subgroups of Lung Carcinoma

Subgroup[a]	Sensitivity (%)	Specificity (%)	PPA (%)	NPA (%)	Accuracy (%)
Left-sided carcinoma	82.2	83.4	63.8	92.2	83.1
Right-sided carcinoma	90.6	88.4	85.3	92.7	89.3
Masses > 3.0 cm	83.6	80.5	63.0	92.5	81.4
Masses ≤ 3.0 cm	70.00	87.5	55.3	93.0	84.3
Squamous	88.6	82.8	70.9	93.9	84.7
Adenocarcinoma	79.5	87.8	68.6	92.7	85.7

[a] No significant differences in sensitivity and specificity were noted between any subgroups (p values ranged between >0.1 and 0.40).

NPA = negative predictive accuracy; PPA = positive predictive accuracy.

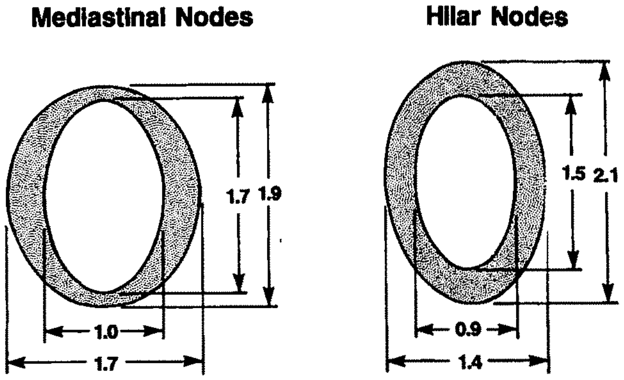
Results

Reliability estimates for prediction of metastatic mediastinal and hilar adenopathy by CT, conventional tomograms, and chest roentgenograms are shown in Table 1. Estimates for CT scan accuracy in the prediction of mediastinal lymph node metastasis for left-sided versus right-sided lesions, masses less than or equal to 3 cm versus larger lesions, and squamous versus adenocarcinoma are shown in Table 2. Similar estimates derived for stages 1, 2, and 3A are shown in Table 3. Computed tomographic scans falsely predicted the absence of mediastinal lymph node metastasis in 19 patients (4.5%). Eleven of these false-negative values occurred in right-sided lesions (57.9%), and eight occurred in left-sided lesions (42.1%). All 19 patients had posterolateral thoracotomies. Because right thoracotomies accounted for 57.7% of the series and left thoracotomies accounted for 42.3%, no side differential for false-negative determinations could be demon-

Table 3. Computed Tomography Reliability Estimates for Detection of Mediastinal Adenopathy in Various Stages of Lung Carcinoma

Stage	Sensitivity (%)	Specificity (%)	PPA (%)	NPA (%)	Accuracy (%)
1	. . .	84.0	. . .	100	84.0
2	. . .	80.4	. . .	100	80.4
3A	77.6	90.9	97.0	51.3	80.4

NPA = negative predictive accuracy; PPA = positive predictive accuracy.


Mediastinal Nodes

Hilar Nodes

Fig 2. Metastatic and noninvolved mediastinal lymph nodes measured at the time of thoracotomy. Shaded areas *represent metastatic lymph nodes. All measurements are in centimeters and reflect mean values.*

strated. All false-negative mediastinal lymph node determinations occurred in patients with stage 3A disease. The mean pathological size measurements for the short axis of these metastatic lymph nodes was only 1.2 cm.

At definitive thoracotomy (n = 364), 69 patients (19.0%) had positive mediastinal nodes and 102 (28.0%) had positive hilar nodes. In intact mediastinal nodes with detailed measurements, 23 of 240 nodes (9.6%) with a short-axis diameter of 1.0 cm or less had metastatic involvement. Long- and short-axis measurements of mediastinal and hilar lymph nodes with and without metastatic disease are shown in Figure 2.

To determine whether the predictive accuracy of CT for mediastinal adenopathy changed with succeeding years of experience or different generations of scanners, we analyzed separately each year between 1979 and 1986. No statistically significant difference in sensitivity, specificity, or overall accuracy could be detected for individual years or type of scanning device used.

When chest CT scans were analyzed for specific components of the TNM system, prediction of all elements of this system was correct in 190 of 418 patients (45.4%). Computed tomography predicted the correct stage but wrong components of the TNM system in an additional 53 patients (12.7%), overestimating the TNM stage in 94 (22.5%) and underestimating the TNM stage in 81 (19.4%). Table 4 compares operative and CT staging.

In 173 patients with detailed pathological and CT measurements of tumor size, the mean tumor diameter determined by CT scanning was 3.5 ± 1.8 cm (range, 1.0 to 12.0 cm). Corresponding pathological measurements were 3.6 ± 1.8 cm (range, 0.7 to 12.0 cm). The CT and pathological sizes agreed within 10% in only 60 of 173 determinations (34.7%), agreed within 25% in 116 (67.0%), and agreed within 50% in 159 (91.9%). Size discrepancies usually resulted from areas of lung collapse or pneumonia adjacent to tumor or contiguity of the neoplasm with surrounding structures.

Computed tomographic scans suggested metastatic disease in liver, lung, adrenal glands, bone, or abdominal lymph nodes in 40 of 373 scans (10.7%). Some patients had specific biopsy of the abnormal organ; others were followed to autopsy or for a minimum of 2 years to ascertain the validity of the finding. Only 5 (12.5%) had documented metastatic disease, resulting in a positive and negative predictive accuracy of 12.5% and 100%, respectively, and an overall accuracy of 90.6%.

Comment

The proper staging of lung cancer requires accurate information about the status of hilar and mediastinal lymph nodes, the relationship of the primary tumor to the major bronchi, mediastinum, and chest wall, and the presence or absence of metastatic disease. Computed tomographic scans are often used to define some of these issues. Can chest CT scans provide accurate information concerning these components?

In 7% to 9.5% of patients [19, present study] with operable lung cancer, tumor may be present in mediastinal lymph nodes 1.0 cm or less in short-axis diameter, producing a false-negative CT scan. In some instances, benign lymph nodes may enlarge to diameters greater than 6 cm secondary to granulomatous disease or anthracosilicosis, thus producing false-positive determinations. Because sensitivity and specificity of an examination reflect all degrees of true and false positivity and negativity, these findings will continue to produce disappointing results when mediastinal and hilar adenopathy is analyzed. In most series, reliability estimates predicting lymph node metastatis average approximately 80% (Table 5). Specific treatment regimens for lung cancer should not

Table 4. Relationship Between Surgical and Computed Tomographic Staging for 418 Lung Cancer Patients

	Stage From CT Scans of Chest and Upper Abdomen				
Surgical Stage	1	2	3A	3B	4
	50.2	5.3	25.8	9.1	9.6
1 (50.7)	36.7[a] (77.6)				
2 (12.2)		2.2[a] (88.9)			
3A (25.6)			13.4[a] (76.8)		
3B (9.6)				4.5[a] (89.5)	
4 (1.9)					1.7[a] (57.1)

[a] These numbers represent percentage of CT scans with correct stage. Numbers in parentheses show percentage of CT scans with all components of correct TNM system for a particular stage.

CT = computed tomography.

Table 5. Reliability Estimates for Computed Tomographic Prediction of Malignant Mediastinal Adenopathy Using Different Size Limits for Lymph Nodes

Author	n	Size Limit	Sensitivity (%)	Specificity (%)	PPA (%)	NPA (%)	Accuracy (%)
Osborne et al [12]	42	0.6 cm	94.4	62.5	65.4	93.8	76.2
Richey et al [14]	48	1.0 cm	95.0	67.9	67.9	95.0	79.2
Khan et al [8]	50	1.0 cm	83.3	90.6	83.3	90.6	88.0
Conte and Bucknam [2]	75	1.0 cm	85.0	89.1	73.9	94.2	88.0
Patterson et al [13]	84	1.0 cm	71.0	88.7	78.6	83.9	82.1
Lewis et al [9]	418	1.0 cm	84.4	84.1	68.7	92.9	84.2
Glazer et al [5]	49	100 mm^2	95.2	64.3	66.6	94.7	77.6
Baron et al [1]	94	1.5 cm	74.4	98.2	96.7	84.4	88.3
Daly et al [3]	338	1.5 cm	79.3	87.9	67.7	93.0	85.8
Ferguson et al [4]	61	2.0 cm	95.0	83.3	79.2	96.1	88.0

NPA = negative predictive accuracy; PPA = positive predictive accuracy.

be based on figures in this range. Positive and negative predictive accuracies reflect relationships between true-positive and false-positive and between true-negative and false-negative determinations, respectively. Because the ratio of true-positive to false-positive determinations is poor with CT scans, the finding of an enlarged lymph node with this method carries little predictive value. The ratio of true-negative to false-negative values is generally high, however, producing a negative predictive accuracy that is usually greater than 90%. This compares favorably with the negative predictive accuracy of various invasive staging procedures [6]. In our institution, potentially operable patients with lung cancer undergo thoracotomy directly if mediastinal lymph nodes are 1.0 cm or less by CT criteria. Tumor present in this subset of patients is usually subcapsular and amenable to a lymph node dissection with a chance of eventual cure. Patients with enlarged lymph nodes by radiologic imaging techniques are subjected to operative staging before definitive decision is made concerning resectability. Results of our series were in agreement with others [7, 10, 12] that conventional tomography and plain chest roentgenograms were unreliable in predicting mediastinal and hilar lymph node metastasis.

Although mean CT and pathological size determinations of pulmonary tumors were close, individual values often varied markedly. In some instances, postobstructive pneumonia or proximity to surrounding structures obscured actual tumor dimension by CT. Computed tomographic determinations of mass size were within 10% of pathological measurements in 34.7% of patients and within 50% in 91.9%. When the tumor was totally surrounded by normal lung tissue, CT assessment of size was very accurate.

Because structures from the thoracic inlet to the level of the adrenal glands are imaged, CT can potentially detect abnormalities within structures other than the lung and mediastinum. In a series of 330 patients with lung cancer [20], 32 adrenal masses (9.7%) were found by CT examination. Eight of 330 (2.4%) represented documented metastasis, and 17 proved to be adenomas. In a series of 172 patients with lung cancer, Pagani [21] reported 20 enlarged adrenal glands (11.6%). Although 6 of 20 patients were presumed to have metastatic disease due to widespread cancer, the remaining 14 had biopsy-proven malignancy. If the remaining 152 patients represented true-negative values, the sensitivity, specificity, and predictive accuracies of this study were 100% each. A similar study [22] of adrenal metastasis documented a sensitivity of 86%, a specificity of 97%, and an accuracy of 93%. In our series, CT suggested metastatic disease outside the involved lung and lymph node drainage paths in 40 of 373 scans (10.7%). In some patients, specific biopsy was performed; others were followed to autopsy or for a minimum of 2 years with repeat images to confirm or refute metastasis. Only 5 of 40 (12.5%) had proven metastatic disease. This yield is quite low and may reflect inclusion of small hepatic cysts and adrenal thickening as metastatic disease in the early part of this series. Decisions concerning possible metastatic disease suggested by CT should be governed by the clinical situation. With rare exceptions, CT abnormalities cannot be considered absolute proof of metastasis. Definitive biopsy or interval scanning must be used to verify these findings. A high negative predictive accuracy for metastases in the chest and upper abdomen makes CT a useful tool for their exclusion.

Can the prediction of mediastinal lymph node metastasis by CT be improved by altering size criteria for abnormality? Receiver operating characteristic curves from two series [5, 23] show distinct cutoff points for mediastinal adenopathy at 1.0 to 1.2 cm. Kiyono and colleagues [24] measured mediastinal lymph nodes at various locations in 40 cadavers. Normal lymph nodes throughout the mediastinum averaged 1 ± 0.2 cm. These studies confirm a cutoff point of 1 cm for estimation of mediastinal lymph node metastasis. Receiver operating characteristic curves for both hilar and mediastinal lymph node metastasis from our series show a flatter slope compared with results of previous studies [5, 23]. By altering size criteria for abnormality, the false-positive determinations could be reduced, but at the cost of lowering the true-positive values. From our curves, no size criteria could be found that would increase the overall accuracy of these scans.

Because false-negative CT determinations and negative predictive accuracies are relatively constant in most series, reduction of false-positive values may be the only means to improve CT accuracy. Buy and co-workers [23] mapped mediastinal lymph nodes according to the expected drainage patterns for lung cancer in a series of 97 patients. Node sizes in other thoracic regions usually not associated with lymphatic spread also were quantified. Lymph nodes were considered abnormal if the short axis of the largest node in the expected drainage pattern was greater than or equal to 1.0 cm and the difference between this node and the largest gland in a noninvolved area was greater than 0.5 cm. By incorporating this dimension in size quantification, positive and negative predictive values were 95 and 94%, respectively, with an overall accuracy of 94%. Unless the positive predictive accuracy can be improved to 100% consistently, however, enlarged mediastinal lymph nodes must be biopsied to exclude metastasis.

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DISCUSSION

DR BENEDICT D. T. DALY (Boston, MA): The accurate staging of lung cancer is becoming increasingly important as the efficacy of preoperative and postoperative neoadjuvant and adjuvant therapy is being evaluated, and it is particularly important in patients with advanced disease. The subject of the paper of Lewis and colleagues is quite timely in this context. They have shown that CT is not accurate in staging lung cancer in patients with positive T, N, or M findings, but that it has proved useful in patient management.

Most previous studies have focused on CT assessment of the mediastinal lymph nodes because prognosis roughly parallels nodal involvement. In the present series of 418 patients, a negative pedictive accuracy of 93% was achieved. The 77% of patients with a negative CT scan immediately underwent thoracotomy without an invasive staging procedure. Lewis and colleagues further make the point that the negative predictive accuracy has not changed over time despite improvement in scanning technology and greater variation in reader interpretation.

We have had a similar experience. In our series, which now numbers 576 patients, the negative predictive accuracy of CT for staging the mediastinal lymph nodes is 92% and has varied little over the years within the range of 92% to 94%. This value has remained relatively constant despite a progressively more aggressive intraoperative evaluation of mediastinal lymph nodes. Between 1986 and 1989, complete subcarinal or paratracheal lymph node dissections, or both, were performed in 47% of our patients rather than routine mediastinal inspection and mediastinal lymph node sampling. Even in this subset of patients, the negative predictive accuracy has remained greater than 90%.

Dr Lewis, how do you evaluate the mediastinum intraoperatively?

In the manuscript, Dr Lewis has provided detailed information regarding the technique of CT scanning used by his colleagues. The hilus is scanned first as contrast medium is being injected. This technique is not routinely necessary to evaluate the mediastinal lymph nodes, and only 9% of our patients received contrast medium; however, contrast medium is required for assessment of hilar adenopathy.

Although you have presented us with the percentages of false-positive and false-negative results for CT staging of the hilar nodes, do you find that information helpful in patient management?

One of the continuing controversies regarding interpretation of CT scans is the size criterion used for evaluating lymph nodes. The receiver operating characteristic curves provided in this series are a sound basis for establishing standard-size criteria for hilar and mediastinal adenopathy. If CT is used as a staging tool in prospective multicenter studies, this standardization is necessary.

Although CT evaluation of the tumor is, as you have shown, inaccurate (and we agree), in what way has it been useful in your experience?

We have also been disappointed in the usefulness of CT in evaluation of adrenal metastases. As Lewis and colleagues have indirectly suggested, a better imaging modality is required.

Finally, although CT cannot be a substitute for definitive surgical pathological staging, it has proved useful in obviating prethoracotomy staging in most patients. Dr Lewis, what percentage of patients undergoing thoracotomy in the present series had resection? Our own figure is 95%, and we believe CT has helped us achieve this.

DR THOMAS W. SHIELDS (Chicago, IL): This controversy has been going on for almost 10 years and will do so as long as we continue to use terms inappropriately. A CT examination is neither positive nor negative. It shows either an enlarged node or a nonenlarged node, and I believe this causes much of the difficulty. As Lewis and colleagues have pointed out, the only real value of CT is that it determines whether a node is less than 1 cm. If this is true, no further preoperative staging is needed. One can perform an exploratory operation. An incidence of 5% or 10% of positive nodes of normal size, such as Lewis and colleagues had in their patients, is, I believe, an acceptable risk. But to discuss whether an enlarged node is positive, negative, or false makes little difference. If a node is enlarged, you must examine it preoperatively.

With regard to staging below the abdomen, the 12.5% figure is midleading because when that number is divided into the number that were positive or suspicious, the percentage is only 1.3%, and this method truly is not cost-effective for imaging the area below the abdomen unless it is part of a standard CT examination of the chest.

This paper was presented very well, but the authors did not show any chest roentgenograms. One of our great losses with increasing use of CT is that we are forgetting how to read a chest roentgenogram, which is the first thing we must learn how to do. The chest roentgenogram should tell us whether a CT scan should be performed. The results of the CT scan will tell us whether we should perform an invasive investigation.

I believe that we really must be very stern at conferences. Inevitably, when a patient is presented, the CT scan is shown first. We must almost beg to see the roentgenogram because it has been lost; that is not the way to perform lung surgery. Computed tomography is of value when it is used properly, but all it determines is whether a node is enlarged or whether it is normal in size. Forget negative or positive.

DR LEWIS: I stage the mediastinum intraoperatively by complete lymph node dissection from the thoracic inlet down the peritracheal area to the subcarinal region and finish at the inferior pulmonary vein on the right side. I perform a similar dissection, including the aortopulmonary window, on the left. This is basically the same kind of dissection that Martini and McCormack have described.

Although the predictive accuracy of CT for hilar lymph node metastasis is low, I still keep it in mind when exploring the chest. I have not found it to be particularly helpful in determining which patients would require, for example, pneumonectomy rather than lobectomy.

Dr Shields, I could not agree more with your comments. I did very quickly show a slide with the staging accuracy figures for plain chest roentgenograms and standard laminography. These two methods have not been very useful in predicting hilar and mediastinal lymph node metastasis. The plain chest roentgenogram, however, can give a better idea of the degree of chronic obstructive pulmonary disease and cardiac compensation, which can affect surgical outcome.

Computed tomographic scanning below the diaphragm is a part of the chest CT determination. This does not incur a separate cost, and the few minutes that are required to obtain it certainly are worth the effort for the information gained.

Surgery for High Cervical Esophageal Carcinoma: Experience With 11 Patients

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The treatment of carcinoma of the cervical esophagus remains controversial. Eleven patients with carcinoma of the high cervical esophagus were encountered over the past 7 years at our institution. There were 6 men and 5 women whose ages ranged from 51 to 72 years. Six patients had tracheal or laryngeal invasion. In all instances one-stage pharyngolaryngoesophagectomy with pharyngogastric (6 patients) or pharyngocolic (5 patients) reconstruction was performed. There was one hospital death. Six patients died 6 to 35 months postoperatively: 1 from recurrence, 2 from generalized metastases, and 3 with both local recurrent and metastatic disease. One other patient died free of disease 6 weeks postoperatively of pneumonia. The remaining 3 patients are alive 12 to 84 months after operation with excellent rehabilitation and good quality of life. We conclude that one-stage surgical resection and reconstruction for high cervical carcinoma of the esophagus offers good palliation and possible long-term survival with acceptable operative risk.

(Ann Thorac Surg 1990;49:597–602)

Cervical esophageal carcinoma has been a vexing problem because of the common advanced stage at presentation, with invasion of important adjacent structures, especially the trachea or larynx, and the difficulty in satisfactorily reconstructing pharyngogastric continuity after extensive resection [1–6]. Recently, multistage procedures with lengthy hospitalizations and high morbidity have been abandoned in favor of one-stage pharyngolaryngoesophagectomy and reconstruction with a suitable graft [2–4, 7–13]. Concomitant laryngectomy has not only allowed more extensive proximal and lateral resection but also has decreased postoperative pulmonary complications associated with reflux and aspiration [13]. Furthermore, complete esophagectomy has been advocated to ensure adequate distal esophageal margins and to treat possible multicentric disease [9, 14]. The stomach [2, 7, 10–12, 14], colon [6, 15], and jejunum [1, 4, 16] have been reported as suitable grafts for reconstruction. Although several investigators [2, 5, 7, 8, 10–12, 14] have reported good results with transhiatal esophagectomy without thoracotomy, transthoracic esophagectomy is safe and is our preferred approach [17].

Although anatomically the cervical esophagus begins at the level of C-6 at the cricopharyngeus muscle and extends approximately 5 cm distally to the level of T-1 at the thoracic inlet [18], most tumors of advanced stage extend beyond these boundaries. In particular, tumors whose proximal extent involves the postcricoid region of the hypopharynx (immediately cephalad to the cricopharyngeus muscle) are difficult to categorize as hypopharyngeal or cervical esophageal cancers. We therefore chose to consider tumors of the upper half of the cervical esophagus that did or did not involve the postcricoid region as high cervical esophageal neoplasms. Because tumors in this region would be expected to behave as cervical esophageal neoplasms [2, 4, 5], we have reviewed our experience for management of patients with high cervical esophageal carcinoma and compared it with other reported series of cervical esophageal cancers.

Material and Methods

From September 1980 to December 1987, 11 patients were treated at Emory University Hospital for high cervical esophageal carcinoma located in the hypopharyngeal or postcricoid region (Table 1). There were 6 men and 5 women with a mean age of 61.7 years (range, 51 to 72 years). All patients had a 1-week to 9-month history of dysphagia (mean, 12.4 weeks). Seven patients had weight loss of 6 to 30 pounds (mean, 20.1 pounds). Two patients had neck soreness, 2 had bleeding, and 1 patient with a tracheoesophageal fistula had intermittent aspiration.

None of the patients had cervical lymphadenopathy on examination. All patients were evaluated with an esophagogram and esophagoscopy. Eight underwent bronchoscopy, and 1 patient had direct laryngoscopy. Thoracoabdominal and cervical computed tomography was performed in 6 patients.

Only 1 patient in the series received preoperative adjuvant chemotherapy. All patients underwent one-stage pharyngolaryngoesophagectomy and reconstruction; 2 patients also had hemitrachectomy. In 6 patients a gastric pull-up with suprahyoid pharyngogastrostomy was performed (Fig 1). In 5 patients a left colon interposition graft was used (Fig 2). Ten of the 11 patients underwent esophagectomy and mediastinal lymph node dissection through a right thoracotomy, and all patients had transverse cervical and upper midline abdominal incisions. One patient underwent transhiatal esophagectomy because of severe pulmonary emphysema, with placement

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Table 1. Patient Data

Initials	Age (yr)	Sex	Symptoms	Tumor Grade/Adjacent Postsurgical Structures Stage^a Involved	Length of Tumor (cm)	Graft Used for Reconstruction	Status	Follow-up (mo)	Postoperative Hospitalization (days)
C.B.	59	M	D	Mod. diff., trachea T4 N0 stage III	2.5	Colon	Dead (−tumor)	1.5	22
P.G.	51	M	D,W	Well-diff., trachea T4 N1 stage III	2.0	Stomach	Alive (−tumor)	72	18
G.R.	68	F	D,W	Well-diff., T2 N0 stage IIa	4.0	Stomach	Alive (−tumor)	84	21
W.I.	64	M	D	Mod./poorly diff., T4 N1 stage III^b	3.0	Stomach	Dead (+tumor)	16	16
R.F.	61	F	D	Mod. diff., T2 N1 stage IIb	5.0	Stomach	Dead (+tumor)	6	65
O.M.	60	F	D,W,B	Mod. diff., trachea, T4 N0 stage III larynx	6.5	Colon	Dead (+tumor)	35	14
J.Y.	72	M	D,W	Well-diff., T4 N1 stage III^b	5.0	Colon (retrosternal)	Dead/hospital	. . .	. . .
B.V.	55	F	D,W,P	Poorly diff., T4 N1 stage III	4.3	Colon	Dead (+tumor)	11	56
R.L.	61	M	D	Mod/poorly diff., trachea, T4 N0 stage III^b	5.0	(1) colon; (2) stomach and jejunum (described in text)	Dead (+tumor)	12	16
J.H.	71	M	D,W,P	Poorly diff., trachea, T4 N0 stage III^b	10.0	Stomach	Dead (+tumor)	10	91
M.O.	57	F	D,W,B,A	Mod. diff., TEF, T4 N0 stage III	5.0	Stomach	Alive (−tumor)	12	31

[a] All patients were stage M0. [b] Lateral margin was involved with tumor on the final pathology specimen.

A = aspiration; B = bleeding; D = dysphagia; diff. = differentiated; mod. = moderately; P = throat pain; TEF = tracheoesophageal fistula; W = weight loss.

of a retrosternal colon interposition graft. In all other patients the bypass graft was placed in the bed of the resected esophagus. Two of the permanent tracheal stomas were mediastinal owing to partial proximal tracheal resection, and 2 other patients underwent concomitant radical neck dissections. In all patients, whenever possible, an attempt at preserving parathyroid function was made by performing only hemithyroidectomy on the side of the lesion and leaving the contralateral side with the inferior thyroid artery intact. Patients with extensive cervical esophageal carcinoma or with recurrent disease, particularly after previous radiotherapy, were at risk of both hypothyroidism and hypoparathyroidism even when the glands were left in situ.

In all patients the tumor was squamous cell carcinoma. The proximal margin of all tumors was located between the postcricoid region and within 2 cm distal to the cricopharyngeus muscle. The tumors ranged in length from 2 to 10 cm (mean, 4.8 cm). Nine patients were in postsurgical stage III, 1 was in stage IIa, and 1 was in stage IIb. In 9 of 11 patients the tumor invaded the periesophageal tissues (T4), and in 2 patients the tumor extended into the muscular layer only (T2). The tumor extended into the posterior wall of the trachea to a varying degree in 6 patients, including 1 patient with a malignant tracheoesophageal fistula. In all patients the proximal and distal margins of resection were free of tumor; however, in 4 patients the lateral margins were positive for tumor microscopically. Five patients had at least one positive regional lymph node, and in 6 patients all nodes were histologically negative, including both patients who underwent radical neck dissections. No patients had known metastatic disease at the time of operation.

Results

One hospital death occurred in the patient with severe emphysema who did not undergo a thoracotomy. The patient had a sudden respiratory arrest on the fourth postoperative day and died 1 week later, never having regained consciousness. Early complications are shown in Table 2; they included one gastropharyngeal fistula that required muscle graft closure. In another patient ischemic colitis required removal of the interposed segment of colon and use of a free jejunal graft between the pharynx and the mobilized stomach (Fig 3). Two patients had respiratory failure secondary to pneumonia (both had gastric pull-up procedures), and in 1 patient a pancreatic abscess developed and required drainage (after a colon interposition procedure). The average length of stay for hospital survivors was 35 days, with 6 patients hospitalized 22 days or less. All patients were maintained initially on either parenteral hyperalimentation or enteral nutritional support through catheter jejunostomy. Oral intake was begun only after the absence of anastomotic leaks was confirmed by oral contrast radiography. Patients

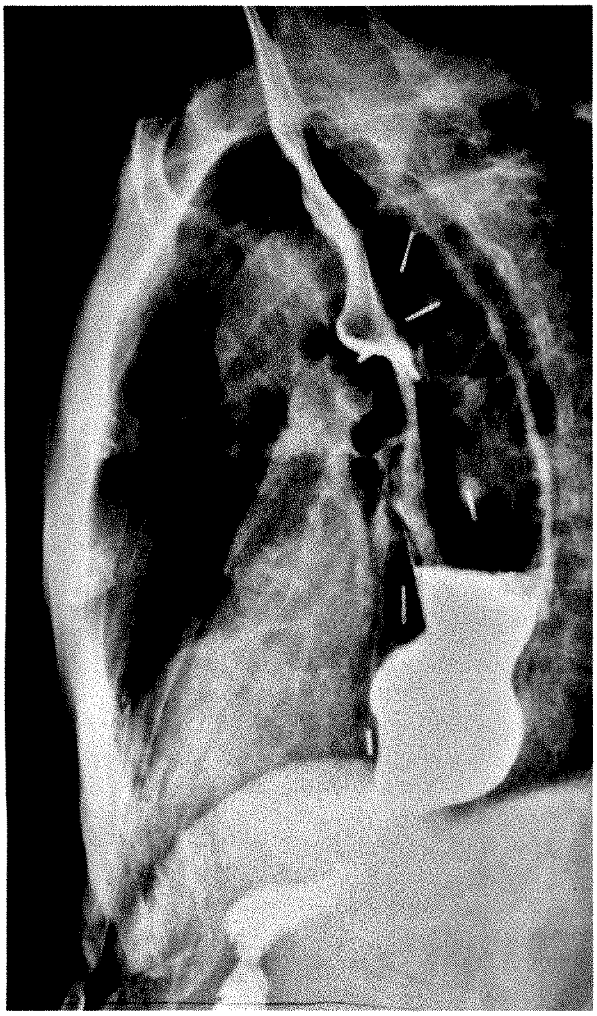

Fig 1. Gastropharyngeal anastomosis after radical laryngopharyngoesophagectomy.

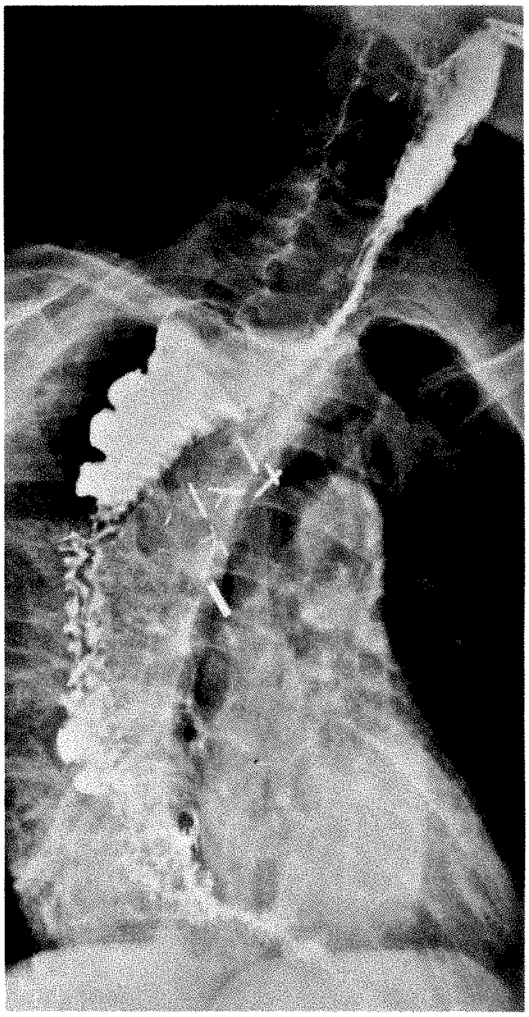

Fig 2. Left colon interposition graft after laryngopharyngoesophagectomy.

were maintained on ventilatory support for a minimum of two days and intensive care unit observation was continued for five to seven days. No patients required supplementary ventilatory or nutritional support at the time of hospital discharge.

Late follow-up of the 10 hospital survivors ranged from 6 weeks to 7 years with a mean follow-up of 24.2 months. There were seven late deaths. Six patients died of their tumor 6 to 35 months after operation (mean, 15 months), and 1 patient died of sudden respiratory distress and ensuing necrotizing pneumonia 6 weeks after operation. No residual tumor was found at autopsy. In patients with tumor-related deaths, 2 had metastatic disease only, 1 had local recurrent disease only, and 3 had both local recurrence and metastatic disease. The mean time to local recurrence was 9.8 months after the original operation. The tumor recurred at the tracheostomy site, at the bypass graft, or at both sites. All 4 patients with local recurrence received radiation therapy.

Three late survivors were alive and free of tumor 7 years, 6 years, and 1 year after operation. In 1 patient (M.O.) late tracheal stomal stenosis required insertion of a permanent tracheostomy tube (Table 2).

All patients were able to subsist totally on oral intake subsequent to surgical reconstruction and had minimal difficulties with reflux. Those patients with local recurrence, development of metastatic disease, or both began to experience difficulty with oral intake before death. Seven of the patients including the late survivors were able to communicate by esophageal speech or with the aid of an electronic device.

Table 2. Early and Late Complications

Complication	Bypass Graft	No. of Patients
Early		
Gastropharyngeal fistula	Stomach	1
Ischemic (graft) colitis	Colon	1
Pneumonia	Stomach (2)	2
Pancreatic abscess	Colon	1
Hypoparathyroidism	. . .	4
Hypothyroidism	. . .	5
Late		
Tracheal stomal stenosis	Stomach	1

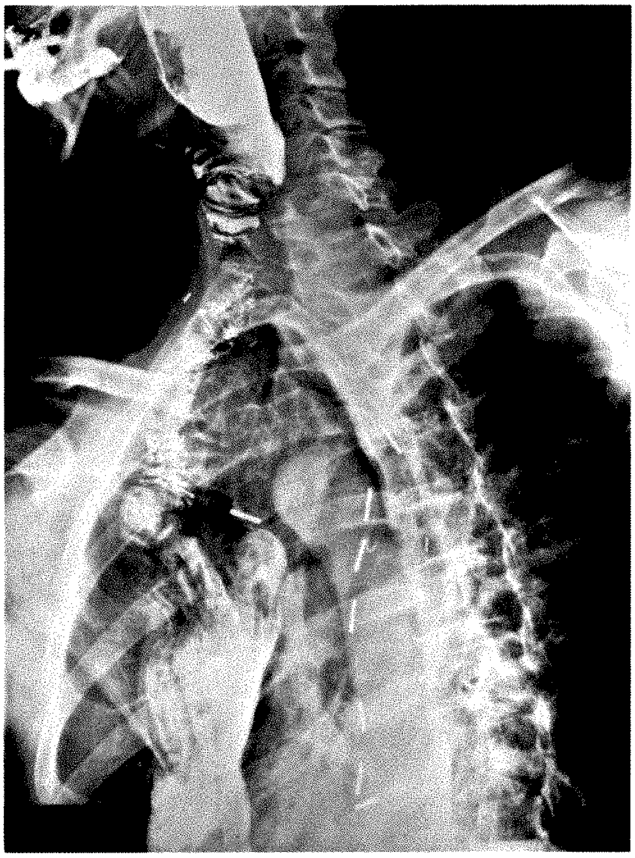

Fig 3. Free jejunal graft between the pharynx and the mobilized stomach (described in text).

Comment

Our series typifies the advanced stage of the disease at presentation [1, 2] with 9 of 11 patients having stage III disease, including 6 patients who had some degree of tracheal or laryngeal invasion. The most advanced degree of tumor invasion occurred in the patient who initially had a symptomatic malignant tracheoesophageal fistula. Because all patients were already seriously symptomatic with dysphagia (100%) and weight loss (64%), we used aggressive surgical management. We performed pharyngolaryngoesophagectomies in all cases to remove all gross tumor, although in 4 cases microscopic lateral margins were positive on final pathological sections. Thus, even with radical resection, extensive local tumor spread does not always allow one to perform a curative procedure [1–3, 12]. On the average the tumors extended 5 cm distally from the postcricoid region, making total cervical esophagectomy a minimum resection. We agree with other investigators [6, 9, 11, 14] that only complete esophagectomy will assure an adequate distal esophageal margin.

Our results confirm the early success of one-stage procedures with only a 9% hospital mortality rate [2, 5, 7, 8, 10, 12]. We have used thoracotomy incisions routinely to facilitate accurate esophageal resection. Our only hospital death was that of a patient with severe respiratory disease who was not believed to be a candidate for thoracotomy and therefore underwent transhiatal esophagectomy.

The length of postoperative hospitalization was comparable with that of other series in which patients underwent one-stage resection [5, 7, 8, 10, 12]. Although the average stay was 35 days, more than half of the patients were discharged approximately 3 weeks postoperatively.

We, like many investigators [2, 5, 8, 11, 12, 18], favor gastric pull-up instead of colon interposition procedures for primary reconstruction owing to the richer blood supply and greater technical ease and lower perioperative and late morbidity. In our series reconstruction was initially performed with stomach (6/11). The left colon (5/11) was used when patients had had a previous gastric operation or when the stomach was unsuitable for bypass. Excluding hypothyroidism and hypoparathyroidism, rates of occurrence of perioperative complications were similar for both groups, with 3 of 6 patients who underwent gastric pull-up procedures and 2 of 5 patients who underwent colon interposition procedures having serious morbidity (Table 2). Furthermore, all patients in follow-up were able to tolerate food without notable reflux regardless of the conduit used.

In our series only 1 patient had graft failure (ischemic colitis); it was not fatal. This complication has been reported to be a serious problem with use of free jejunal graft reconstruction, occurring with a 17% incidence and a 12% related mortality [1]. Although we used this technique of secondary reconstruction in 1 patient, our results indicate that primary reconstruction with colon or stomach may be safer from the standpoint of graft viability and related mortality.

The late results reflect the advanced stage of disease at presentation. Thirty percent of the patients are alive and free of tumor 1 to 7 years after operation. None of these patients received adjuvant therapy. Five of the 6 patients with late tumor-related death were known to have metastatic disease at the time of death and had a mean survival of 11 months. The 1 patient who died with only known local recurrent disease survived 23 months from the time of diagnosis of recurrent disease (35 months postoperatively). Thus, including the 3 late survivors, mean survival in this group without metastatic disease was 46 months. We conclude that aggressive surgical therapy not only improves the quality of life in these severely symptomatic patients but also offers them the opportunity for prolonged survival. Although in some patients metastatic disease will develop, adversely affecting their survival, patients free of metastatic disease may expect a prolonged satisfactory quality of life even in the face of recurrent local disease.

Whether failure of local control, which occurred in 40% (4/10) of our hospital survivors, could have been reduced by the use of routine perioperative adjuvant therapy is uncertain. Because 2 of the 3 hospital survivors with positive microscopic lateral margins had local recurrence, we infer that wider resection margins (if obtainable), adjuvant therapy, or both might help eradicate residual tumor and decrease the rate of local failure. Recent reports [19, 20] have not yet shown a significant improvement in long-term survival with use of preoperative radiation therapy alone in cases of cervical esophageal carcinoma. Although Hilgenberg and colleagues [21] reported a 54% survival at 3 years in patients with squamous cell carcinoma of the thoracic esophagus treated with perioperative chemotherapy and radiation therapy, similar encouraging results have not yet been demonstrated in cases of cervical esophageal cancers.

Attempts at curative treatment of cervical esophageal carcinoma by high-dose radiation therapy alone have resulted in less than 25% 5-year survival and were dependent on tumor size [22–24]. Unfortunately, this modality is associated with both a high incidence of late complications, including carotid artery rupture, esophageal stricture, and tracheoesophageal fistula, and a high incidence of local recurrence of disease (66% in Lee's series) [23]. Furthermore, radiation therapy is contraindicated in the presence of a malignant tracheoesophageal fistula. Finally, except for radiation therapy, other modalities of palliative treatment of cervical esophageal carcinoma, including laser therapy [25] and intubation [26], are unsatisfactory.

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DISCUSSION

DR JOHN R. HANKINS (Baltimore, MD): My colleagues and I at the University of Maryland have done four transhiatal esophagectomies for high cervical esophageal carcinomas. In 2 patients we extended the procedure to include removal of the larynx, in other words, we performed a laryngopharyngoesophagectomy. In the other 2 patients, whose tumors appeared to have partially responded to chemotherapy and did not seem to directly invade the trachea, we shaved, or to state it more accurately, carefully dissected the tumor off this structure. There were no operative or hospital deaths. All 4 patients enjoyed fairly satisfactory deglutition until they died of recurrent cancer 8 to 14 months later.

I have two questions. First, the abstract states that only 6 patients had tracheal or laryngeal invasion and yet you apparently removed the larynx in all 11 patients. What was your indication for removing the larynx in the 5 patients in whom there was not direct invasion? Second, why did you use the transhiatal route in only 1 patient? We find the transhiatal approach much easier to perform and much better tolerated by the patient than the three-incisional approach (abdominal, thoracic, and cervical).

DR MANSOUR: To answer the second question first, I did not use the transhiatal route because I do not believe in that approach. There are many ways to skin a cat. At Emory I never thought that transhiatal or "blunt" esophagectomy is the proper way to perform an operation. I was brought up to believe that operation should be done under direct vision. I do not like that "shaving" of tumors. I like to have wide resection of upper and lower margins and take the whole esophagus through the right side of the chest with the surrounding mediastinal structures. I like to bring the graft into the posterior mediastinum so that it comes behind the permanent tracheostomy and is situated far above the tracheostoma rather than coming across it in the neck.

The first question is why we removed the larynx in all the patients, though the larynx was not involved in all of them. If you resect the esophagus above the cricopharyngeus, you cannot control aspiration at all. In all patients, the larynx was removed and the anastomosis was performed between the pharynx and the stomach or colon to prevent aspiration and its dreaded complications.

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The Thirty-seventh Annual Meeting of the Southern Thoracic Surgical Association will be held at the Hyatt Regency Cerromar Beach, Puerto Rico, November 8–10, 1990. There will be a $155 registration fee for nonmember physicians except for guest speakers, authors and coauthors on the program, and residents. There will be a $50 registration fee for attendees of the Postgraduate Course, which will be held the morning of Thursday, November 8, 1990. The Postgraduate Course will provide in-depth coverage of thoracic surgical topics selected primarily as a means to enhance and broaden the knowledge of practicing thoracic and cardiac surgeons.

Members wishing to participate in the Scientific Program should submit an original abstract and one copy by May 15, 1990, to Donald C. Watson, Jr, MD, Program Chairman, Southern Thoracic Surgical Association, 111 East Wacker Drive, Chicago, IL 60601. Abstracts must be submitted on the Southern Thoracic Surgical Association abstract submission form. These forms may be obtained from the Association's office or in this issue of *The Annals of Thoracic Surgery*. All slides used during the presentation must be 35 mm. Manuscripts of accepted papers must be submitted to *The Annals of Thoracic Surgery* prior to the 1990 meeting or to the Secretary-Treasurer at the opening of the Scientific Session.

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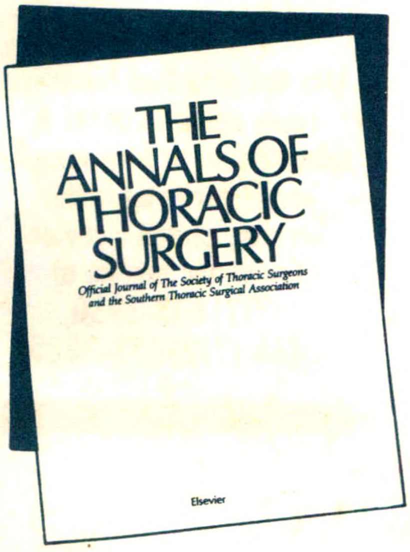
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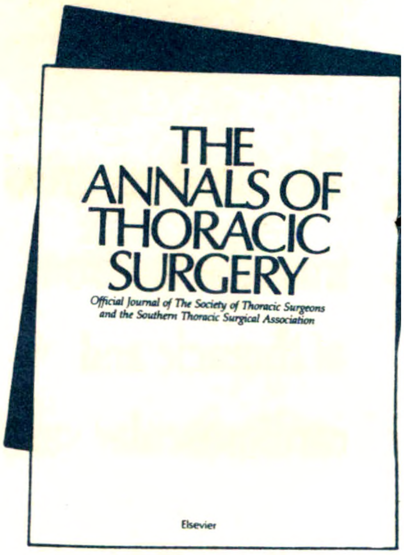
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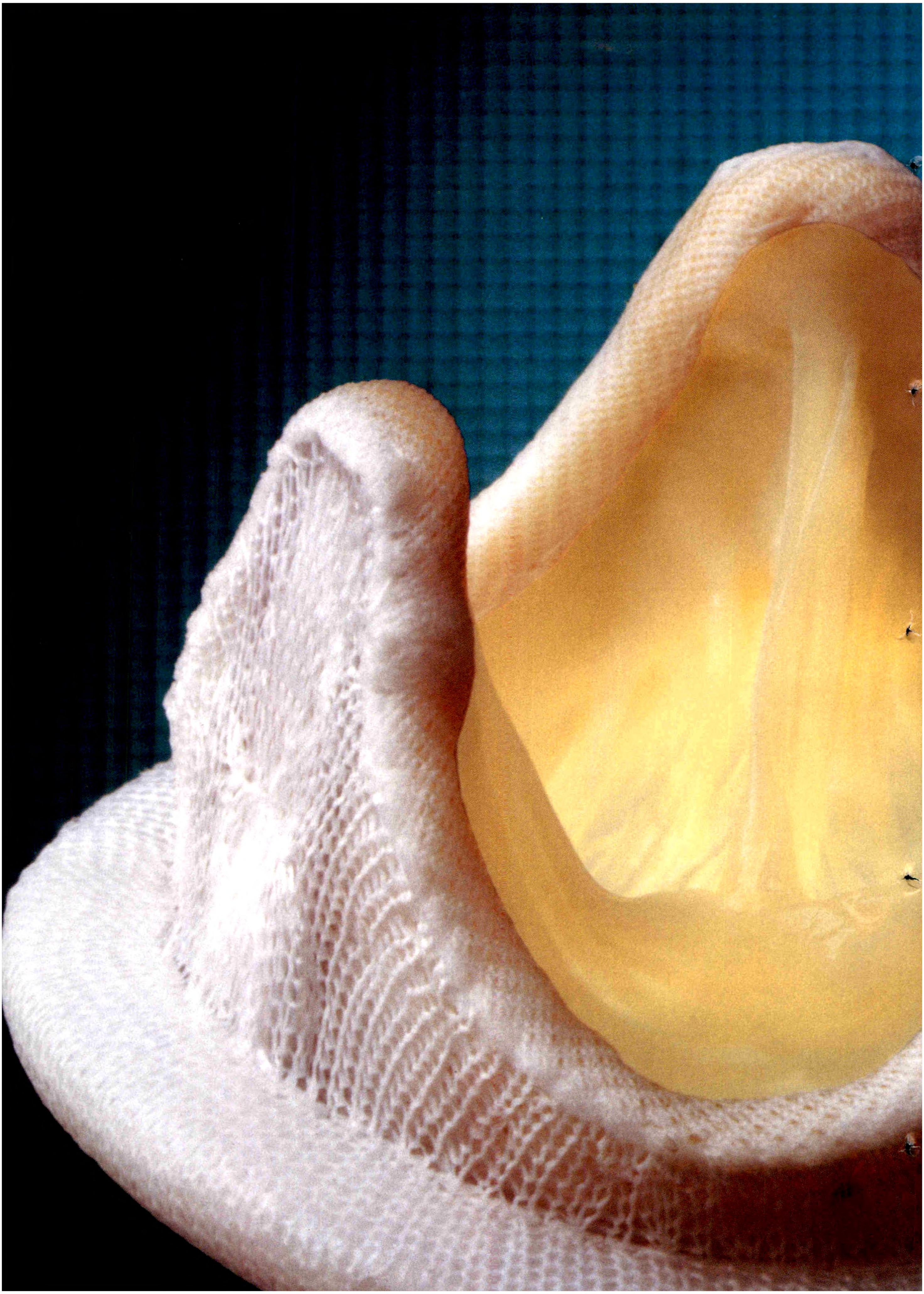

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The decision to use a tissue valve must ultimately be made by the physician on an individual basis after a careful evaluation of the short- and long-term risks and benefits to the patient are made, and alternative methods of treatment are considered. Overall durability, especially long-term, has not been established for bioprostheses. Serious complications, sometimes leading to replacement of the valve and/or death, may be associated with the use of prosthetic valves (see Complications). A full explanation of the benefits and risks should be given to each prospective patient before surgery. **Note:** Bioprostheses should be used with caution in the presence of severe systemic hypertension or when the anticipated patient longevity is longer than the known longevity of the prosthesis. 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The appropriate anticoagulation therapy must be determined by the physician on an individual basis. **Adequate rinsing with physiological saline, as described in the Technique section, is mandatory before implantation to reduce the glutaraldehyde concentration. No other solutions, drugs, chemicals, antibiotics, etc. should ever be added to the glutaraldehyde or rinse solutions, as irreparable damage to the leaflet tissue, which may not be apparent under visual inspection, may result. The Carpentier-Edwards bioprostheses must be kept moist at all times. Drying out will cause irreversible damage to the leaflet tissue. To prevent drying out during implantation, the valves should be irrigated periodically on both sides with sterile physiological saline.** Passing diagnostic catheters and transvenous pacing leads across a Carpentier-Edwards bioprosthesis could damage the valve. Consequently, it is recommended that these procedures be avoided. Both the valve and glutaraldehyde storage solution are **sterile**. The outside of the jar is **not sterile** and must **not** be placed in the sterile field. Furthermore, the valve and container should **never** be subjected to sterilization procedures involving ethylene oxide, propylene oxide, steam, or irradiation. Storage between 10°C and 25°C is recommended; however, care should be exercised to avoid freezing or extreme heat, which may damage the valvular tissue. **Any valve that has been frozen or exposed to temperatures greater than 60°C should not be used for human implantation.** Glutaraldehyde may cause irritation of the skin, eyes, nose, and throat, and may also cause skin sensitization. Avoid prolonged or repeated contact or prolonged breathing of the vapor. Use only with adequate ventilation. In the event of contact, immediately flush the affected area with water. In the event of contact with eyes, seek medical attention. **Surgical Precautions: 1.** When choosing a valve for a given patient, the size, age, and physical condition of the patient in relation to the size of the prosthesis must be taken into consideration to minimize the possibility of obtaining a suboptimal hemodynamic result. The selection of a valve, however, must ultimately be made by the physician on an individual basis after carefully weighing all of the risks and benefits to the patient. **2.** Due to the relative flexibility of the frame, care must be exercised to avoid the use of too large a prosthesis to prevent folding or extreme deformation of the stent that may render the valve incompetent. Oversizing may create highly localized mechanical stresses resulting in tissue failure in the form of detachment of the leaflets or porcine aortic wall from the valve stent posts. **3.** Left ventricular perforation or ulceration has been reported. Both open and closed chest cardiac massage in patients with an open strut mitral prosthesis increases the risk of ventricular perforation. Additionally, lifting, flexing, and manual compression of the distended heart after discontinuation of bypass may cause a commissure strut to be pushed through the endocardium, producing a dissecting hematoma with subsequent uncontrolled hemorrhage. **4.** The spacing of the sutures in the remnant of the valvular orifice and the prosthesis suture ring must be carefully matched to avoid folding of the leaflets or extreme distortion of the orifice. Edwards CVS Division has received reports in which individual mattress sutures, spanning a distance of 10 to 15mm, produced a purse-string effect causing compression of the valve orifice. **5.** When using interrupted sutures, it is important to cut the sutures close to the knots and to ensure that exposed suture tails will not come into contact with the leaflet tissue. Cases have been reported in which bioprostheses developed severe regurgitation and had to be replaced as a result of wear due to contact with sutures. **6.** Unlike rigid mechanical valves, the stent wall is soft and will not resist needle penetration. Accordingly, extreme care must be exercised when placing sutures through the sewing margin to avoid penetration of the side wall of the stent and possible laceration of the leaflet tissue. **7.** As with all prostheses that have open cages, free struts, or commissure supports, care must be exercised, particularly in mitral replacement, to avoid looping or catching a suture around the free legs which would interfere with proper valvular function. **8.** The stent of the bioprosthesis is asymmetrical to conform to the natural shape of the porcine aortic valve. Consequently, the commissure supports are not equidistantly spaced. The contrasting suture markers in the sewing ring of the mitral valve denote the greatest intercommissural distance. These two commissure supports should be oriented so as to straddle the ventricular outflow tract to avoid murmurs due to blood flow turbulence. **Note:** The contrasting suture material should not be removed from the prosthesis. **9.** In the aortic position, the struts should correspond to the remnants of the natural commissures so as not to obstruct the coronary ostia. **10.** A serial number tag is attached to the sewing ring of each valve by a suture. This serial number should be checked against the number on the jar; if any difference is noted, the valve should be returned unused. This tag should not be detached from the valve until implant is imminent. Care should be exercised to avoid cutting or tearing the suture ring cloth during removal. **11.** Gentle handling is required for all implantable devices. If the valve is dropped, damaged, or mishandled in any way, it must not be used for human implantation. **Complications:** As with all prosthetic heart valves, serious complications, sometimes leading to death, may be associated with the use of tissue valves. In addition, complications due to individual patient reaction to an implanted device, or to physical or chemical changes in the components, particularly those of biological origin, may occur at varying intervals (hours or days) necessitating reoperation and replacement of the prosthetic device. Complications associated with the use of Carpentier-Edwards bioprostheses include stenosis, regurgitation through an incompetent valve, perivalvular leak, endocarditis, hemolysis, thromboembolism, thrombotic obstruction, bleeding diatheses related to the use of anticoagulant therapy, ventricular perforation by stent posts, looped sutures, and malfunctions of the valve due to distortion at implant or physical or chemical deterioration of valve components. Types of tissue deterioration include infection, calcification, thickening, perforation, suture abrasion, instrument trauma, and leaflet or aortic wall detachment from the valve stent posts. These complications may present clinically as abnormal heart murmur, shortness of breath, exercise intolerance, dyspnea, orthopnea, anemia, fever, arrythmia, hemorrhage, transient ischemic attack, stroke, paralysis, low cardiac output, pulmonary edema, congestive heart failure, cardiac failure, and myocardial infarct. **Note:** Based on reports in the literature on tissue valves, there appears to be an increased incidence of leaflet calcification in patients under the age of 20. In this regard, animal research studies show that a high systemic calcium level can lead to early calcification. Furthermore, at least one published report describes an apparent causal relationship between the consumption of daily calcium supplements and early leaflet calcification in an adult. When feasible, repeated intravenous injections containing calcium should be avoided during the postoperative period; and excessive milk or dairy product consumption should be avoided in children. 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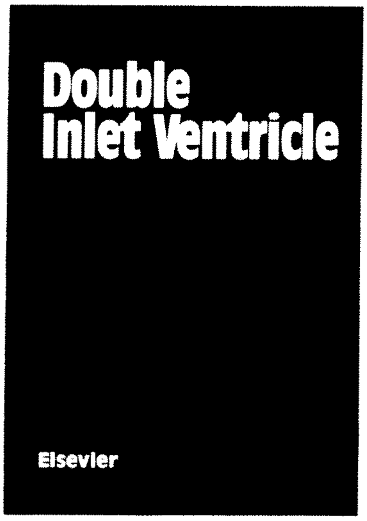
A complete and concise review



Edited by
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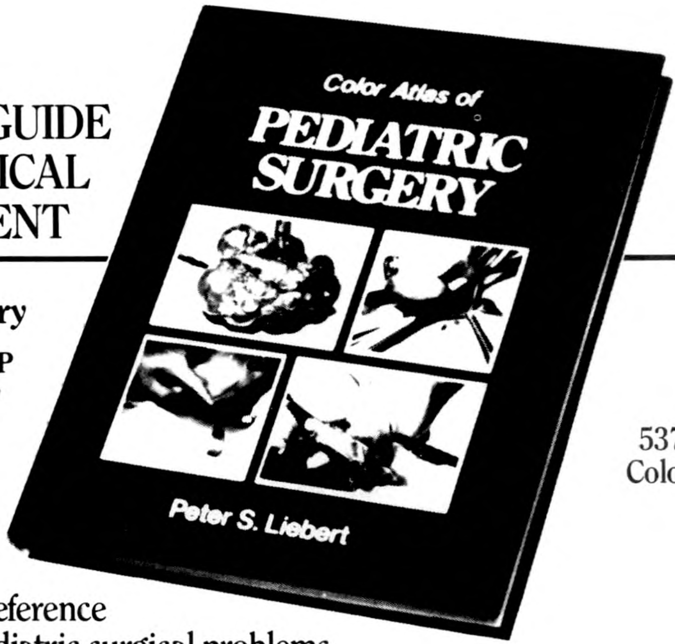
College of Physicians and Surgeons
New York



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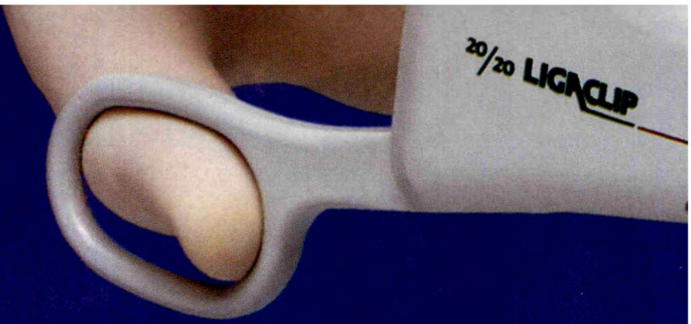

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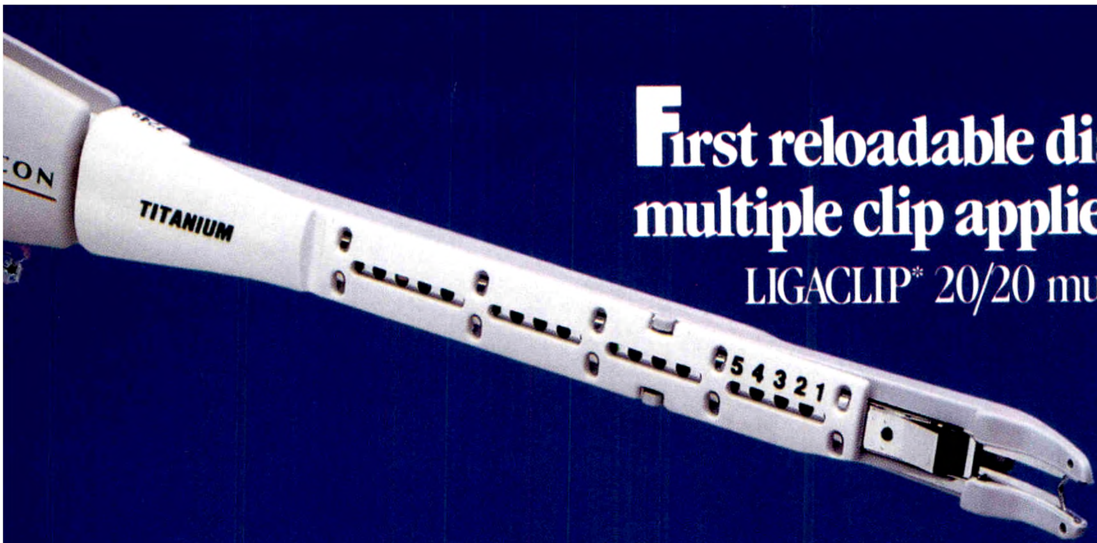

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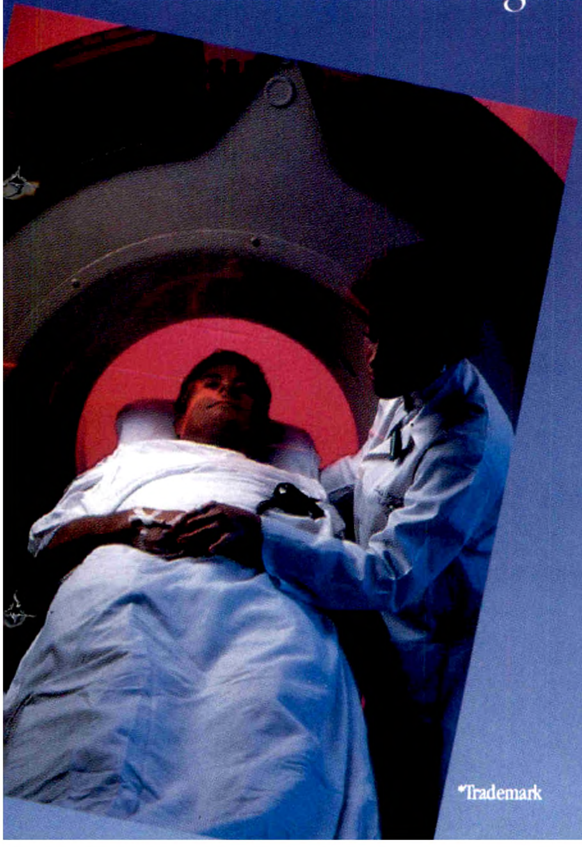

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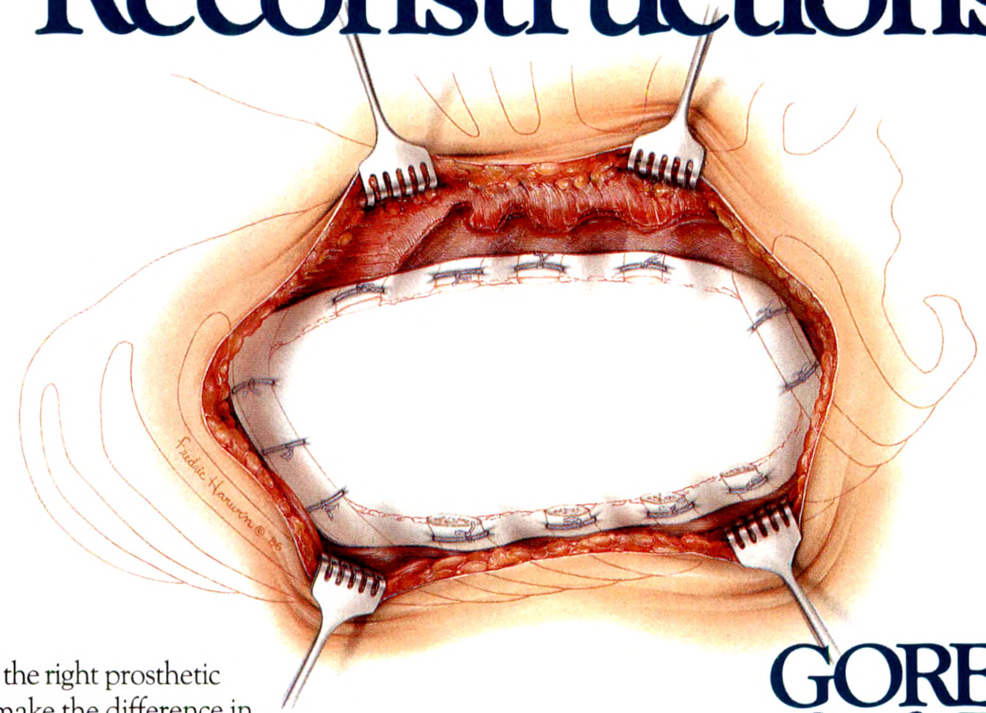

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2. Brown GL, et al. Comparison of prosthetic materials for abdominal wall reconstruction in the presence of contamination and infection. Annals of Surgery 201(6):705-711, 1985.

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True Emergency Coronary Artery Bypass Surgery

Fred H. Edwards, MD, Ronald F. Bellamy, MD, J. Robert Burge, MS,
Amram Cohen, MD, LeNardo Thompson, MD, Michael J. Barry, MD, and
Lawrence Weston, MD

Department of Cardiothoracic Surgery, Walter Reed Army Medical Center, Washington, DC, and the F. Edward Hebert School of Medicine, Uniformed Services University of Health Sciences, Bethesda, Maryland

Previous reports of emergency coronary artery bypass grafting often included cases that were not true surgical emergencies, thereby creating inappropriately favorable results. To accurately investigate this important subgroup of patients, we analyzed our recent experience with truly emergent coronary artery bypass grafting. From January 1984 to January 1989, 117 patients underwent true emergency bypass grafting for acute refractory coronary artery ischemia. Clinical deterioration was associated with failure of percutaneous angioplasty in 37 patients and instability during diagnostic catheterization in 13 patients. Refractory ischemia developed in the remaining patients while on the ward or in the intensive care unit. All operations were performed within four hours of surgical consultation, most within one hour. Overall in-hospital operative mortality was 14.5% (17/117), and 76.5% of deaths (13/17) were due to cardiac-related causes. Major morbidity occurred in 35.9% (42/117). Univariate analysis isolated ejection fraction, extent of coronary artery disease, previous myocardial infarction, hypertension, need for inotropic support, use of an intraaortic balloon pump, and cardiopulmonary resuscitation as risk factors for operative mortality. Stepwise multivariate analysis confirmed that previous myocardial infarction, hypertension, cardiopulmonary resuscitation, and reoperation were independently significant risk factors. Age, sex, diabetes, left main disease, and peripheral vascular disease had no significant impact on the prognosis. The 4% operative mortality (2/50) for patients taken directly to the operating room from the catheterization suite was significantly lower than the 22.4% mortality (15/67) associated with emergencies arising on the ward or intensive care unit ($p < 0.01$). A logistic risk equation developed from this population accurately modeled operative mortality. All 9 patients predicted to have a greater than 90% risk of operative death died, whereas only 1 (1.5%) of the 65 patients predicted to have a less than 5% risk died. We conclude that true emergency coronary artery bypass grafting carries a particularly high risk, with an operative mortality in the range of 15%. This finding merits full consideration when making therapeutic decisions concerning this group of critically ill patients.

(Ann Thorac Surg 1990;49:603–11)

In recent years, emergency coronary artery bypass grafting (CABG) has become an important part of most cardiac surgery programs. The operative mortality for emergency CABG has been the subject of several reports leading to a general impression that surgical revascularization in this setting can be carried out with only moderate risk. There is considerable evidence to indicate that surgeons can anticipate an operative mortality in the range of 5% for such patients.

A close review of previously published reports indicates that the patient population studied by many groups may not have direct relevance to current surgical practice. In several instances, some portion of the population was made up of patients who did not represent true surgical emergencies. In other reports, patients requiring emergency CABG reoperation were arbitrarily excluded even though such patients are not uncommon. Some of the most favorable results were reported during the early days of percutaneous transfemoral coronary angioplasty (PTCA) when multivessel angioplasty was seldom attempted and PTCA for unstable ischemia was infrequent.

Given the increased number of patients seen for emergency CABG, it is quite important to have a firm appreciation of the attendant operative risks. Accordingly, we analyzed our experience with truly emergent CABG, including reoperations, in the era of aggressive coronary angioplasty.

Material and Methods

In 1984 we developed an aggressive approach to acute coronary artery ischemia. Our general philosophy was directed to obtain prompt revascularization of compromised vessels using thrombolytic therapy, PTCA, CABG, or some combination of these modalities. Patients who failed to obtain adequate symptomatic resolution from intravenous administration of nitroglycerin were evaluated by our cardiology service and were treated with thrombolytic agents or PTCA, or both, or were directly referred for emergency CABG. All patients having refractory ischemia after unsuccessful thrombolytic therapy or PTCA were referred for emergency CABG.

Presented at the Thirty-sixth Annual Meeting of the Southern Thoracic Surgical Association, Scottsdale, AZ, Nov 9–11, 1989.

Address reprint requests to Dr Edwards, Department of Thoracic Surgery, Walter Reed Army Medical Center, Washington, DC 20307–5001.

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We were careful to include in this series only those patients with coronary ischemia that was refractory to all other appropriate forms of therapy. Those patients having ischemic events that resolved by the time of surgical consultation were not included even though we often performed CABG within an hour of the initial event. For example, patients found to have critical left main coronary artery disease with successful pharmacological treatment of ischemia were not included, nor were those having a single episode of ischemia that resolved with intravenous nitroglycerin and intraaortic balloon pump (IABP) placement. Even though such patients usually underwent revascularization right away, they were not included in this report. All patients in this series had either clinical or electrocardiographic evidence of ongoing, medically refractory coronary artery ischemia at the time of consultation.

In general, we used an IABP preoperatively whenever it could be placed promptly by our cardiologists. If the condition of the patient was deteriorating rapidly, however, we opted to immediately move to the operating suite because of our ability to quickly institute cardiopulmonary bypass. Likewise, if the IABP could not be placed easily, we invariably proceeded directly to the operating room rather than risk further delay.

Some patients with refractory ischemia were either not referred by the cardiologists or were not offered CABG by our surgeons. These patients were judged to be unacceptable surgical candidates because of uncontrolled cancer, dementia, serious new neurological deficits, other incapacitating medical maladies, or the absence of coronary vessels amenable to CABG. In addition, those patients whose ischemia was caused by a coronary vessel supplying only a small amount of myocardium were not offered surgical revascularization.

From January 1984 to January 1989, 117 patients satisfying our criteria underwent emergency CABG at our hospital. The time from the surgical consultation to the initial incision in the operating room was less than four hours in all instances and was usually less than one hour. All patients taken to the operating room from the catheterization suite had the initial incision made within one hour of consultation. The clinical profile of these patients is shown in Table 1. The average age was 59.4 years (range, 30 to 80 years).

Sixty-seven patients (57%) were taken to the operating room from the intensive care unit (ICU). The condition of the remaining 50 patients deteriorated in the catheterization suite after PTCA (37 patients) or diagnostic catheterization (13 patients), and they were taken to the operating room directly from the catheterization laboratory.

Operations were performed using cardiopulmonary bypass with moderate systemic hypothermia and topical cooling. Cold potassium crystalloid or blood cardioplegia was used in all patients. The distal anastomoses were performed initially, and the proximal anastomoses were constructed over a partial occlusion clamp during rewarming. Our general approach was to perform the first anastomosis to the vessel thought to be most responsible for acute clinical deterioration. The average number of vessels bypassed was 2.8, with a minimum of one (17 patients) and a maximum of five (6 patients).

Table 1. Patient Characteristics[a]

Variable	Survivors (n = 100)	Non-survivors (n = 17)	Total (n = 117)
Age (yr)			
<50	20 (20)	3 (18)	23 (20)
50–70	65 (65)	11 (65)	76 (65)
>70	15 (15)	3 (18)	18 (15)
Sex			
Male	74 (74)	16 (94)	90 (77)
Female	26 (26)	1 (6)	27 (23)
Ejection fraction[b]			
<0.30	4 (5)	3 (19)	7 (7)
0.30–0.50	18 (20)	5 (31)	23 (22)
>0.50	67 (75)	8 (50)	75 (71)
Coronary artery involvement			
Left main	21 (21)	4 (24)	25 (21)
3 Vessels	52 (52)	14 (82)	66 (56)
2 Vessels	31 (31)	3 (18)	34 (29)
1 Vessel	17 (17)	0 (0)	17 (15)
Previous MI	38 (38)	13 (77)	51 (44)
Reoperation	8 (8)	5 (29)	13 (11)
Cardiogenic shock	2 (2)	6 (35)	8 (7)
IABP	25 (25)	6 (35)	31 (27)
Diabetes	9 (9)	3 (18)	12 (10)
PVD	13 (13)	5 (29)	18 (15)
Hypertension	30 (30)	10 (59)	40 (34)

[a] Numbers in parentheses are percentages. [b] The ejection fraction was determined from ventriculography in 105 patients. In the remaining 12 patients, ventriculography was precluded by clinical deterioration or renal failure.

IABP = intraaortic balloon pump; MI = myocardial infarction; PVD = peripheral vascular disease.

If the patient was young and in hemodynamically stable condition with no adverse electrocardiographic changes at the time cardiopulmonary bypass was instituted, we considered using the internal mammary artery (IMA) as a conduit. The left IMA was taken down during cardiopulmonary bypass in 16 patients and was used to bypass the left anterior descending coronary artery in each instance.

A statistical analysis of the risk factors listed in Appendix 1 was carried out to determine those variables that were associated with an adverse outcome. All calculations were made using a standard statistical program (BMDP Statistical Software, Los Angeles, CA). A p value of less than 0.05 was considered significant. Univariate analysis of each risk factor was tested by calculating a χ^2 value with 2 × 2 contingency tables and 1 degree of freedom. If the number of events or the sample size was small, Fisher's exact test was used. The multivariate analysis was carried out using stepwise logistic regression to determine those variables having an independent impact on patient survival. Those factors found to be of significance in the multivariate analysis were then used to generate a logistic

Table 2. Postoperative Morbidity[a,b]

Category	No. of Patients
Low-output syndrome	16 (14)
Atrial fibrillation	9 (8)
Respiratory insufficiency	6 (5)
Reoperation for bleeding	6 (5)
Sternal dehiscence	3 (3)
Perioperative MI	11 (9)
Acute renal failure	9 (8)
Neurological deficit	5 (4)
Extremity ischemia	3 (3)
Minor wound infection	4 (3)

[a] Refer to Appendix 3 for definitions. [b] Numbers in parentheses are percentages.

MI = myocardial infarction.

risk equation to model patient outcome as a function of preoperative risk factors (Appendix 2).

Results

Operative Mortality

Seventeen (14.5%) of the 117 patients died at some point during the hospitalization in which CABG was performed. Five died in the operating room, 2 died on the first postoperative day, and only 1 died more than 30 days after CABG. The cause of death was cardiac related in 13 patients (76.5%). Two of the other 4 patients died of multisystem failure, 1 died of sequelae of hemorrhagic gastritis, and 1 with good postoperative cardiac function sustained irreversible brain damage.

Of the 37 patients taken directly to the operating room after a PTCA misadventure, only 1 (2.7%) failed to survive. There were 13 additional patients who were taken directly to the operating room from the catheterization suite after diagnostic coronary angiography complicated by coronary artery dissection, thrombosis, or embolization, and there was 1 nonsurvivor (7.7%) in this group as well. The overall operative mortality for the 50 patients taken directly from catheterization to CABG was 4% (2/50). The remaining 67 patients taken to operation from the ward or ICU made up the majority of operative deaths with a 22.4% operative mortality (15/67).

Operative Morbidity

Major morbidity occurred in 35.9% of patients (42/117) and is broken down by category in Table 2. All three instances of distal extremity ischemia were due to an IABP placed in the common femoral artery, and none of the 3 patients required amputation or fasciotomy, although thrombectomy was necessary in 1. Thirty-one IABPs were placed preoperatively, and an additional 23 were placed in the operating room, for a total of 54 in this series. Complications directly attributable to the IABP occurred in 5.6% of these patients (3/54). A left ventricular assist device was used in 2 patients, both of whom died.

Three of the 9 patients having acute renal insufficiency survived, and all recovered normal function. Neurological problems were manifest in 5 patients and consisted of confusion or delirium in 3, a transient ischemic attack in 1, and a massive cerebral infarction in 1. Four of the 6 patients requiring reexploration for bleeding survived, and all of the patients who had sternal dehiscence survived.

Risk Factor Analysis

Both univariate analysis and the multivariate analysis of preoperative patient variables were carried out to determine which factors were significantly ($p < 0.05$) correlated with operative mortality. In the univariate analysis, ejection fraction, number of coronary arteries involved, previous myocardial infarction (MI), hypertension, need for inotropic support, need for an IABP, and cardiopulmonary resuscitation (CPR) were identified as significant risk factors for operative mortality. When patients representing a PTCA emergency were considered as a separate category from those with a diagnostic catheterization emergency, the statistical analysis failed to demonstrate a significant difference in operative mortality compared with those patients taken to CABG from the ward or ICU. When all patients representing emergencies in the catheterization suite are grouped together, however, there is a significant difference ($p < 0.01$) in operative mortality compared with those patients going to operation from elsewhere in the hospital.

Stepwise multivariate logistic regression showed that previous MI, CPR, hypertension, and reoperation were independently significant risk factors. Age, sex, diabetes, left main coronary artery disease, and peripheral vascular disease had no significant impact on the prognosis.

None of the 17 patients with single-vessel coronary artery disease died, whereas 21.2% (14/66) of those with triple-vessel disease died. Left main coronary artery disease carried an operative mortality of 16.0% (4/25). All of the nonsurvivors in this study had some compromise of the left anterior descending coronary artery, and although it was sometimes impossible to unequivocally implicate this vessel as being responsible for the acute deterioration, we believe it played a major role in most of the deaths.

There were 13 patients (11.1%) requiring emergency CABG after previous coronary artery operations, and the operative mortality in this group was 38.5% (5/13). None of these 13 patients had undergone more than one previous CABG procedure. Two of the 13 were in cardiogenic shock as they were being taken to the operating room, and both died. One of the 13 was a septuagenarian, and he also died. Two additional patients undergoing reoperation required CPR en route to the operating room, and 1 survived.

Use of the IMA was not associated with an increase in complications. There was no significant difference between the 12.5% operative mortality (2/16) for the IMA group and the 14.9% operative mortality (15/101) for those revascularized with saphenous veins only. In 1 of the 16 IMA patients, electrocardiographic changes indicative of ischemia developed while the vessel was being taken

Table 3. Predicted Versus Observed Operative Mortality

Predicted Mortality (%)	Observed Mortality[a] (%)	95% Confidence Limits[b]
<5	1.5 (1/65)	0%–9.3%
5–25	8.8 (3/34)	2.3%–24.8%
25–50	0.0 (0/1)	. . .
50–75	50.0 (4/8)	17.4%–82.6%
75–100	100.0 (9/9)	62.9%–99.0%

[a] Within the parentheses, the numerator is the number of deaths and the denominator is the total number of patients for that risk category. [b] The confidence limits indicate that one can be 95% certain that the "true" observed mortality will lie within the specified interval.

down, and this individual had a perioperative MI. There was no significant difference in the rate of perioperative MI. In the saphenous vein group there were 10 perioperative MIs (10%), and in the IMA group, one MI (6.3%).

Risk Stratification

To assign patients into appropriate risk categories, a logistic regression model was developed. The four risk factors found to be significant predictors of operative mortality by multivariate analysis were selected as predictor variables and were used to develop an equation of the form

$$P = 1/[1 + \exp(-X)],$$

where $X = B_0X_0 + B_1X_1 + \ldots B_kX_k$, P is the probability of postoperative death, the X values are predictor variables, and the B values are derived weights for those variables. Appendix 2 lists the B values for this patient population.

To test the model, the predictor variables for each patient were entered into the risk equation, and a predicted probability of operative death was calculated for each of the 117 patients. The predicted results were then compared against actual operative results in two ways. Table 3 shows the results obtained when patients are categorized into arbitrary risk groups. There is reasonable agreement, particularly at each end of the risk spectrum. A more direct way to assess the risk equation is to use it as an absolute predictor of operative mortality. Using this approach, if P is greater than 50%, then the test is positive, predicting that the patient will die. If P is less than 50%, the test is negative, and the patient is predicted to survive. Using this method, the accuracy of the test was 94.9%, with 76.5% sensitivity and 98% specificity (Table 4).

Comment

Early experiences with emergency CABG were associated with a poor salvage rate [1]. The initial pessimism from these reports was largely reversed a decade later by the pioneering efforts of Berg and colleagues [2, 3] in Spokane and Phillips and associates [4] in Des Moines. These groups advocated a particularly aggressive surgical approach to acute coronary ischemia and established emergency CABG as a viable therapeutic alternative. As the concept of emergency CABG evolved, there were words of caution along the way [5–7], but the precedent had been set for this relatively new mode of treatment to become a well-accepted option for select patients. Several groups have confirmed the feasibility of emergency CABG in recent years, and operative mortality rates of less than 8% are often reported [2–4, 8–10]. In a recent summary of treatment options for acute coronary ischemia [11], it was flatly stated that emergency CABG should be carried out with an operative mortality of less than 5%.

Our experience indicates that this may be an important misconception. Many previous reports were based on patient populations not wholly comprised of true surgical emergencies, whereas other reports covered a time period in which aggressive, multivessel PTCA was not common. Our results, along with those of others [12–14], lead us to the conclusion that true emergency CABG in the current era of PTCA carries an operative mortality of approximately 15%.

Review of Previous Reports

It is not our intent to criticize previous reports that have led to the widespread application of this essential therapeutic advance. We fully acknowledge the major contribution of each of these groups, but we believe that there are differences in the patient populations that must be emphasized.

Our purpose in this study has been to focus on the risks associated with emergency CABG in patients having acute coronary artery ischemia refractory to all other appropriate modes of therapy. This is a particularly important subset of patients that has not been completely examined in the current practice of cardiac surgery. Patients in this situation require immediate revascularization to minimize myocardial damage.

In the series reported by Phillips and colleagues [4] in 1986, some patients did not undergo CABG for up to 36 hours after the onset of ischemia. In addition, some of the patients in whom PTCA failed were taken to operation on the basis of compelling anatomy rather than physiological compromise, and patients having reoperation were arbitrarily excluded. The overall operative mortality was 5.7% among the 261 patients who received surgical reperfu-

Table 4. Confidence Limits of Selected Indices

Index	90% Confidence Level	70% Confidence Level
Accuracy (95%)	90%–98%	92%–97%
Sensitivity (77%)	54%–91%	61%–88%
Specificity (98%)	93%–99%	95%–99%
Predictive value of		
Positive test (87%)	64%–97%	72%–95%
Negative test (96%)	91%–99%	93%–98%

sion. Our approach to acute coronary ischemia closely parallels the clinical algorithm followed by the Iowa Heart Center, and we wholeheartedly agree with their philosophy in this setting, but some patients they have included in their series would not have been included in the population we presented here.

DeWood and associates [3] reported an enormous experience with surgical treatment of acute MI, but a large portion of high-risk patients were excluded from consideration [9]. Less than one third of these patients had triple-vessel disease, and only 15% had sustained a previous MI. Some patients in this population underwent operation as much as 24 hours after clinical deterioration. In a more recent report from the same group, they [2] confirm the feasibility of surgical treatment of evolving MI based on a 2.9% operative mortality (30 days). A number of patients in that series were in sufficiently stable condition to allow CABG up to 12 hours after the onset of ischemia, and those having reoperation were not included. Again, we applaud the excellent results obtained with this challenging group of patients, but the population presented in our study is quite different.

Guyton and co-workers [8] recently reported a 4.4% operative mortality, which is one of the most successful series in the literature. In this study, however, the concept of an emergency operation was not based on physiological compromise, but was defined as an operation that was not scheduled at the beginning of the surgical day at 7:30 AM.

Flameng and colleagues [9] described their experience with emergency CABG in 48 patients having evolving MI. It appears that all the patients in this series experienced refractory ischemia necessitating revascularization within two to four hours after the onset of symptoms. The operative mortality was 8.3% overall. However, there is no mention of the time frame over which this series was gathered. None of the patients required reoperation. Selection criteria were not discussed, thus making it difficult to determine which patients were offered CABG and which were not considered surgical candidates.

The Cleveland Clinic experience with emergency operation after failed PTCA was reviewed by Golding and co-authors [10] in 1986. They reported an operative mortality of 2.5% among 81 patients. Some procedures were performed up to 24 hours after the acute PTCA failure, and no reoperations were included. Left ventricular impairment was absent or mild in 85% of patients, and more than half had single-vessel coronary artery disease. Only 11% had triple-vessel disease.

Athanasuleas and associates [12] from Birmingham reported the results in 83 patients undergoing emergency CABG for evolving MI. It appears that these patients had refractory ischemia in spite of all other modes of appropriate treatment, thereby leaving CABG as the only feasible means of reperfusion. Patients having reoperation were not included, but otherwise this series closely approximates the type of patients in our population. The operative mortality was 15.6% overall and 5.9% in patients less than 65 years of age without cardiogenic shock.

Parsonnet and colleagues [13] examined their experience with CABG after failed PTCA and found that emergency operation carried an 11.9% operative mortality. Certainly most of the patients in this series appeared to require immediate surgical revascularization, but the definition of failed PTCA included only those with angiographic evidence of vessel occlusion. Such patients would not be included in our population unless there was associated ischemia refractory to other forms of treatment. Reoperations were not mentioned in the series.

A similar report from the Mid America Heart Institute [14] presented an operative mortality (30 days) of 11.3% for emergency CABG after failed PTCA. Patients having reoperation were considered in this series, but some underwent CABG more than 24 hours after the failed angioplasty.

Naunheim and associates [15] from St. Louis have exhaustively investigated their CABG experience in recent years, and they provide a particularly valuable insight into emergency operation associated with the contemporary practice of aggressive, multivessel angioplasty. They have demonstrated that the current group of PTCA candidates has a relatively higher risk than those of the early 1980s and as a result, patients requiring emergency CABG after failed PTCA are at higher risk. In their series, 11% of patients died after emergency CABG for failed PTCA. Their population of 103 patients included 12 who underwent revascularization within the first 24 hours after PTCA, and 1 who had CABG four days later. There was no mention of patients requiring reoperative CABG.

Risk Factors in Emergency CABG

We found that previous MI, hypertension, need for CPR, and reoperation were independently significant risk factors for operative death. Several other investigators [8, 12, 15, 16] have found elderly patients to be at higher risk, but age was not significant in our population. Other commonly described risk factors in this setting are cardiogenic shock [9, 12, 14, 15] and compromised ventricular function [12, 15–17]. In addition, reoperation has been described as a predictor of operative death in the only other series that specifically addressed such patients [14]. Killen [14], Naunheim [15], and their co-workers indicate that multivessel disease is an important risk factor for patients requiring emergency CABG after failed PTCA, and several groups [8, 14, 15] emphasize that the current spectrum of patients undergoing emergency CABG after failed PTCA are at higher risk compared with 5 to 6 years ago.

A number of other factors play a probable, but less well defined role in the operative outcome of these patients. The presence of coronary collaterals logically should afford some protection during an acute event, and some groups [12] place considerable emphasis on this finding. Phillips and co-authors [4] described a characteristic mottled, irregular endocardium as angiographic evidence of irreversible myocardial damage and proposed that reperfusion therapy is of no benefit when such findings are present.

The amount of time elapsed from acute clinical deterioration until reperfusion has been discussed in several reports. There appears to be little conclusive evidence that this interval plays a major role in clinical practice. Laboratory studies are only marginally applicable because of the inability to create a model that could account for collateral flow, pharmacological interventions, and mechanical interventions, all of which can come into play in treating a patient with acute coronary ischemia. Several groups [2, 3, 9] believe that time elapsing between onset of symptoms and reperfusion is paramount and will determine the extent of residual infarction; others [4, 12] have placed a lesser emphasis on its importance. Recognizing that the issue is unsettled, our approach was to obtain surgical reperfusion as quickly as possible regardless of the amount of time that had elapsed from the onset of symptoms.

Over the period of this study, our cardiologists have become increasingly persistent in attempting to correct PTCA complications. Acute coronary occlusion is often treated with repeat balloon dilation or thrombolytic therapy, and coronary dissections are sometimes "tacked up" by other balloon procedures. Implantable stents are available in some centers, but have not yet been used in our hospital. Certainly the successful application of these corrective techniques is gratifying, but when these measures fail, the patient may well have sustained myocardial injury that adds to the risk of CABG. Most surgical reports encourage prompt emergency CABG after failure of PTCA [4, 8, 14, 15], although at least one group [18] considers the percutaneous corrective procedures to be worthwhile.

We found that patients whose condition began to deteriorate in the catheterization suite fared significantly better than those in whom ischemia developed on the ward or in the ICU. A χ^2 analysis of these populations reveals several important differences: the group coming from the ward or ICU had more patients greater than 70 years of age ($p < 0.05$), fewer patients with an ejection fraction greater than 0.50 ($p < 0.05$), more patients with triple-vessel disease ($p < 0.001$), and fewer patients with single-vessel disease ($p < 0.001$). In addition, all 5 patients requiring CPR came from the ward or ICU. Furthermore, immediate treatment was rendered in the catheterization laboratory, whereas some delay was inherent in recognizing and treating such problems elsewhere in the hospital.

There is most probably a significant difference in the risks associated with unstable angina compared with those associated with an evolving MI. However, it is often quite difficult to accurately differentiate between the two [4, 19]. Although this may merit consideration in other reports, we have not seen the need to specifically address it here, as all of our patients had unrelenting ischemia and the syndrome producing that ischemia was not of practical importance.

Risk Models

Risk models allow one to mathematically consolidate the impact of preoperative risk factors to predict the probability of operative death for an individual patient. Models are derived from the known operative experience of a patient survey, and in general, models derived from a large number of patients will be more reliable than those created from a smaller series. Under ideal circumstances, validation of the model is carried out by prospectively testing the model against another group of patients for whom the outcome is not known.

In this series we used a logistic risk equation to serve as a model of operative mortality. The number of patients in this study is fairly small, so to obtain the maximum possible reliability, we considered it necessary to use all 117 patients to derive the model, thereby leaving none available for prospective evaluation. Validation of the model was then necessarily carried out by testing the model against the same patients used in the derivation. This is not optimal, but it is not without precedent [20] and is the only possible means of validation when dealing with a small sample size.

Given this restriction, Tables 3 and 4 indicate that the risk equation afforded reasonable accuracy in modeling this series. It was particularly reliable at the extremes of risk. All 9 patients predicted to have a greater than 75% risk of operative death died, whereas only 1 (1.5%) of the 65 patients predicted to have a less than 5% risk died.

We encourage the use of modeling techniques as an essential element of risk analysis. Univariate and multivariate analyses can identify the most significant risk factors for a patient population, but in practice, it is difficult to apply that information to an individual patient. With risk models, it is possible to obtain an estimate of risk for an individual patient based on the previous documented experience of an institution. In addition, such models also allow interinstitutional comparisons or direct comparisons against an accepted standard of care [21].

Application in Clinical Practice

We did not identify a subgroup of patients presenting a prohibitive risk for emergency CABG. Two patients requiring reoperation were in shock as they were taken to the operating room, and both died. On the other hand, 2 other patients requiring reoperation had CPR in progress in the operating room, and 1 survived. The only septuagenarian having reoperation in this series died, but no conclusions can be drawn from a single patient.

Guyton [8], Naunheim [15], and their associates believe that octogenarians with postinfarction cardiogenic shock are not candidates for emergency CABG. We agree that this probably represents a sound approach, but we have not encountered such patients in this study.

Patients less than 70 years of age who were not in cardiogenic shock and did not require reoperation had an operative mortality of 7.4% (6/81). This is quite similar to the results of Athanasuleas and co-workers [12], which leads us to the conclusion that patients in this subgroup can undergo emergency CABG with only moderate risk.

Limitations of This Study

As with all retrospective studies, conclusions must be cautiously applied in clinical practice. Patients seen with acute coronary ischemia are a heterogeneous group because of the spectrum of associated diseases, the variations of coronary compromise, and the possible interventions that may have taken place before surgical consultation. Clearly a prospective, randomized trial would be helpful in this area, but there is some doubt that such a study is truly feasible [12]. Recently, a randomized, controlled study of surgical reperfusion for evolving MI was presented by the University of Ottawa [22], but patients greater than 65 years of age were excluded, as were those with previous infarctions and those in shock.

The medical management of acute coronary ischemia can vary from one institution to another. Our cardiologists have taken an aggressive approach to these patients with the goal of obtaining reperfusion in all patients except those already specifically described. Centers having different exclusion criteria may not be able to extrapolate the results of this series to their own practice.

The definitions used to define risk factors vary considerably from one institution to another. We have listed the arbitrary definitions used in this study in Appendix 3 and believe that these are reasonably well accepted. If future studies are to allow valid comparisons between institutions, there should be some uniformity in this area. It is hoped that some consensus can be reached in regard to the precise definitions of these important terms, including the definition of an "emergency" case.

We should also point out that our approach to these patients has undergone some changes over the time covered in this study. In 1984 our cardiologists were considerably more cautious in using PTCA for multivessel disease and acute ischemia. As experience was gained, patients began to undergo more challenging PTCA procedures. This served to slightly reduce the total number of emergency CABG procedures with time, but it also made those patients requiring emergency CABG a higher-risk group [8, 14, 15]. Although the small number of patients in this series disallows firm statistical verification of this impression, we do believe that there has been some change in the spectrum of patients seen for emergency operation from 1984 to 1989.

Conclusions

Surgical consultation to evaluate patients with acute coronary artery ischemia is becoming more common. An operative mortality of 3% to 6% can be obtained if patients having reoperation are excluded and if one includes patients in sufficiently stable condition to allow revascularization 12 to 24 hours after clinical deterioration. Within this patient population lies a particularly important subgroup that has not been specifically addressed in the current era of aggressive multivessel angioplasty. In this study, we excluded those patients in whom CABG could be safely delayed for 12 to 24 hours to focus on those having acute refractory ischemic syndromes requiring prompt revascularization. Patients in this category constitute true surgical emergencies.

In previous studies from this institution, we [21] have established that our operative results with coronary artery procedures are in keeping with accepted standards of care. Nevertheless, our operative mortality for patients requiring true emergency CABG is considerably higher than the 3% to 6% reported by others. Our results, as well as those from other groups [12, 13] addressing a similar population, indicate that an operative mortality in the range of 12% to 15% can be anticipated from the surgical management of patients representing true emergencies. In current clinical practice, this is a realistic estimate of operative risk and deserves full consideration when making therapeutic decisions for these desperately ill patients.

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Appendix 1. Preoperative Patient Risk Factors

1. Age
2. Sex
3. Myocardial infarction (either old or in progress)
4. Ejection fraction
5. Left ventricular end-diastolic pressure
6. Left main coronary artery involvement
7. Extent of coronary artery disease
8. Presence of ventricular aneurysm
9. Intravenous nitroglycerin
10. Intravenous inotropic agents
11. Intraaortic balloon pump
12. Cardiopulmonary resuscitation
13. Reoperation
14. Angioplasty failure
15. Coronary dissection
16. Presence of hypertension
17. Presence of diabetes
18. Presence of peripheral vascular disease

Appendix 2. Constants for Logistic Regression Equation

$B_0 = -4.4738$	$X_0 = 1$
$B_1 = 5.4648$	X_1 = Myocardial infarction (MI) in progress
$B_2 = -25.9910$	X_2 = MI within the previous 24 hours
$B_3 = 0.4711$	X_3 = MI within the previous week
$B_4 = -25.6770$	X_4 = MI more than a week ago
$B_5 = 4.4385$	X_5 = Need for cardiopulmonary resuscitation
$B_6 = 2.2539$	X_6 = Hypertension
$B_7 = 2.5894$	X_7 = Reoperation

If the factor associated with X is present in a given patient, the corresponding value of X is 1. If the factor is not present, then X is 0 for that factor.

Appendix 3. Definitions

Extent of coronary artery disease. Catheterization data were used to determine the presence of left main coronary artery disease (>50% compromise of vessel diameter) and the number of other major coronary artery vessels involved (>70% narrowing in any view).

Ejection fraction was determined from ventriculography or, in some cases, from nuclear medicine studies that had been done previously.

Coronary artery dissection was recorded if an iatrogenic dissection occurred during either percutaneous angioplasty or diagnostic catheterization.

Patients were considered to have had *angioplasty failure* if unsuccessful angioplasty had been performed at any time during the hospitalization.

Previous myocardial infarction was defined as electrocardiographic evidence of a transmural infarction. Those patients having angina with persistent refractory ST segment elevation or new Q waves were considered to have an *infarction in progress.*

Cardiogenic shock was defined as a clinical state of hypoperfusion characterized by a systolic pressure of less than 80 mm Hg or a cardiac index of less than 2.0 L/min/m^2.

Supportive measures. The use of intravenous nitroglycerin, inotropic support, or the intraaortic balloon pump was recorded as a risk factor only if required for hemodynamic stability or control of ischemia. Dopamine hydrochloride used in low doses only for enhancement of renal perfusion was not included.

Peripheral vascular disease was considered present if there was a history of extremity claudication, rest pain, stroke, transient ischemic attacks, reversible ischemic neurological deficits, or previous extracardiac vascular procedures.

Diabetes mellitus was recorded as a risk factor if any form of medication was required for control.

Perioperative myocardial infarction was listed as a complication for those patients having new Q waves and levels of the myocardial-specific isoenzyme of creatine kinase greater than 5% of total.

Low-output syndrome refers to the need for inotropic support to maintain adequate peripheral perfusion.

Respiratory insufficiency was considered present if the patient required ventilator support for more than 48 hours after operation.

Acute renal failure was recorded if the serum creatinine level was more than 1 mg/dL greater than baseline postoperatively.

Minor wound infections were defined as wound infections not accompanied by sepsis and requiring only incision and drainage under local anesthesia.

DISCUSSION

DR HARVEY W. BENDER, JR (Nashville, TN): Dr Edwards, would you clarify your use of the term *reoperation*? Were you talking about patients who were in an emergency situation and subsequently underwent operation? You also mentioned postoperative reexploration. Would you relate that to the mortality?

DR EDWARDS: Patients described as having a reoperation had undergone coronary bypass grafting in the remote past. In this series, all those requiring reoperation had undergone myocardial revascularization once before. On the other hand, postoperative reexploration refers to those patients whose emergency operation was complicated by the fact that they had to be returned to the operating room because of unacceptable postoperative bleeding.

DR BENDER: How many of those patients died?

DR EDWARDS: We lost 2 of the 6 patients taken back to the operating room for bleeding.

DR GEORGE C. KAISER (St. Louis, MO): This is an important paper and was very nicely presented. Our definitions of emergency and urgent operations are slightly different from yours. We classify anyone who is in unstable condition and who requires intravenous heparin, intravenous nitroglycerin, intravenous β blockade, or a combination of these as urgent. We reserve the term *emergency* for patients with hemodynamic instability or ongoing ischemia. We do not use the 24-hour limit. If you define this group in this manner, as you have indicated, then the mortality rate is quite high for these patients. The results will depend on how an emergency operation is defined.

Part of the problem probably has arisen from the fact that when the Coronary Artery Surgery Study (CASS) group divided their patients into the various operative categories, those investigators used time until operation. This was necessary because CASS was a large, multicenter study, and this definite standard could be applied to the many cooperating centers. Those investigators used 24 hours or less as the criterion for calling an operation an emergency. As you have shown, this is not really an appropriate definition.

DR EDWARDS: Thank you, Dr Kaiser. It really is all a matter of definitions, isn't it? I agree that the CASS group has perpetuated the concept that the term *emergency* includes all those patients operated on within 24 hours of a clinical event. Certainly the majority of thoracic surgeons recognize that as an inappropriately liberal definition of an emergency. In fact, that is one of the major reasons we looked into the subgroup I presented today. This subgroup is an important population because we see these true emergencies frequently, and we have to recognize the relatively high mortality associated with their surgical revascularization.

DR GEORGE R. DAICOFF (St. Petersburg, FL): There is another big variable here, even when comparing one patient with another in one institution, let alone different institutions, and that is how aggressive the medical management is before you are called. To say it is an emergency four hours within the time you get a consultation is not exactly fair.

DR EDWARDS: I think that is exactly right. We considered these patients emergencies because they had ongoing, refractory ischemia, not because they had operation within four hours of consultation. In fact, we often operated within an hour or two on patients with critical anatomy or a single episode of ischemia, but we did not include those patients in this series unless they also had unrelenting coronary ischemia.

The subject of time deserves specific mention. We considered several alternatives to use as the start point and the end point of the time interval to CABG. Some people use the time of clinical deterioration as the start point. We have had a hard time doing that at our institution because often the patients will have a stuttering pattern of ischemia and we cannot really pinpoint when the clinical event arose. Should you start the clock at the first event or the most important event? What should we use as the end point? Is that the time the patient arrives in the operating room, when we make the initial incision, when we get the patient on the pump, or when we complete the revascularization? I think your point emphasizes that there are a lot of variables that need to be ironed out, and a well-controlled clinical trial is probably the only way it can be adequately handled.

DR JOSEPH M. CRAVER (Atlanta, GA): One of the problems that perhaps prompts truly emergency cases is the absence of intercoronary collaterals to compensate for an acute closure of another coronary vessel that would precipitate a crash. In light of this, we have been particularly impressed with how effective retrograde cardioplegia through the coronary sinuses is in getting effective protection by the cardioplegic solution to the myocardium distal to the total occlusion. In light of this experience and your relatively high mortality, have you modified your myocardial protection program, particularly in patients with poor ventricular function or those requiring reoperation, in an effort to reduce both your mortality and perioperative infarction rates, which are high?

DR EDWARDS: In the time frame I presented here, we had not changed over to retrograde cardioplegia. Since then, we have just begun to use it. I think in the situation you described in a patient who does not have a rich network of collaterals, it would be very advantageous.

DR MEREDITH L. SCOTT (Orlando, FL): I thank Dr Edwards and associates for their honesty. I think we ought to have a category of resurrection. That category should record how many survived, not how many died. I think that that last resuscitation group is another category. It is not a class 4; it is a class 5.

DR EDWARDS: I appreciate your point. One of the disappointments we had in this study was that we were not able to find a group who represented a prohibitive risk. We had hoped to find a subgroup of patients about whom we could say, "No, we should not operate on these patients." However, we were not able to do so.

Surgical Treatment of Cardiac Tumors: A 25-Year Experience

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From 1964 to 1989, we performed operations on 133 patients with cardiac tumors. There were 58 male and 75 female patients ranging in age from three days to 81 years; 101 were adults, and 32 were children (less than 12 years of age). Primary tumors (102 benign and 12 malignant) were found in 114 patients and metastatic tumors in 19. Symptoms included congestive heart failure, arrhythmias, emboli, and chest pain. Diagnosis was accomplished through angiography, echocardiography, computed tomography, and magnetic resonance imaging. Operative treatment encompassed techniques ranging from biopsy to complete excision (including hypothermic circulatory arrest and cardiac autotransplantation) depending on the site of disease and the extent of involvement. Overall operative survival was 91%. Twelve patients died early (within 30 days of operation), and follow-up was obtained for 110 (90.9%) of the remaining 121 survivors (total patient-years of follow-up, 572.8; mean follow-up, 5.2 years). Of the 20 patients who died late, 15 had malignant disease. Operative survival for patients with primary cardiac malignancies and for those with metastatic disease was 83% and 68.4%, respectively, with 3 and 5 patients, respectively, still living. We advocate an aggressive surgical approach, especially in patients with benign tumors, who can expect an excellent outcome. For patients with malignant or metastatic disease, palliation and cure are also possible if aggressive surgical actions are taken.

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Cardiac tumors are rare, with an incidence ranging from 0.0017% to 0.03% at postmortem examination in different series [1–3]. Much has changed since Hodgkin first distinguished tumor from thrombus in the early 19th century and since Crafoord first reported successful excision of a cardiac tumor using cardiopulmonary bypass in 1955 [4].

In the 25-year period 1964 to 1989, we saw 133 patients with cardiac neoplasms. This report reviews our experience with numerous cardiac tumors, both benign and malignant, and primary and metastatic. Our data suggest that an aggressive surgical approach is generally safe, usually efficacious, and appropriately considered as therapy for otherwise untreatable lesions.

Material and Methods

We reviewed the hospital records and follow-up clinical charts of 133 patients with heart tumors who were treated at our institution. Of these patients, 114 had primary tumors (102 were benign, and 12 were malignant), and 19 had metastatic tumors. There were 101 adults and 32 children (less than 12 years old), and the age range was three days to 81 years. We analyzed our patients by placing them in groups according to the type of tumor and reviewed the presenting symptoms, diagnosis, and surgical treatment.

Benign Tumors

MYXOMA. The most common benign tumor in this series was myxoma, affecting 55% of the patients, all of whom were adults (Table 1). Three patients had previously undergone resection of a myxoma, had local recurrences, and were referred to us for further treatment. Presenting symptoms included congestive heart failure (44 patients), palpitations (14), emboli (13), constitutional symptoms (11), syncope (10), and chest pain (8). Five patients had no symptoms.

Diagnosis was made preoperatively by angiography in 47 patients. It revealed intracavitary filling defects (fixed or mobile), neovascularity, and valvular obstruction or insufficiency. Pulmonary hypertension (mean pulmonary artery pressure greater than 20 mm Hg) was present in 27 of 39 patients in whom intracardiac pressures were recorded. Echocardiography confirmed the diagnosis in 35 patients and was the sole method used in 4 of them. Diagnosis was made during operation for suspected mitral valve disease in 5 patients. Tumors were located in the left atrium in the majority of patients. There were no tumors in either ventricle.

Currently, we completely excise myxomas through a biatrial approach during total cardiopulmonary bypass support under moderate hypothermia (30°C) with bicaval cannulation for venous drainage. The full thickness of the septum is excised if the lesion is in the region of the fossa

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Table 1. *Profiles of Patients With Primary Benign Tumors*

		Sex		Age		Deaths		Follow-up		
Tumor Type	No. of Patients	M	F	Range	Mean	Early[a]	Late	No. of Patients	Range	Mean (yr)
Myxoma	63	24	39	21–81 yr	51 yr	2	3 (2[b])	50[c]	2 mo–25 yr	7.1
Purkinje cell	13	4	9	2–20 mo	12.6 mo	0	1	12	6 mo–8 yr	3.8
Rhabdomyoma	9	2	7	3 days–1 yr	5.3 mo	1	0	8	7 mo–8.5 yr	3.1
Fibroma	7	3	4	1 mo–12 yr	3.3 yr	0	0	7	2–18 yr	7.3
Lipoma	4	2	2	21–65 yr	49 yr	0	0	4	7–13 yr	8
Other	6	3	3	9–55 yr	39.1 yr	1	1[b]	5	2–22 yr	7.1
Total	102	38	64	3 days–81 yr	36.17 yr	4	5 (3[b])	86	2 mo–25 yr	6.5

[a] Early death is defined as death within the perioperative period or within 30 days of operation. [b] These deaths were unrelated to the tumor. [c] Of the 61 patients who lived more than 30 days after operation, 50 were followed up and data were available.

ovalis. The atrial septal defect created is closed with a Dacron patch or, less commonly, by primary closure [5].

PURKINJE CELL. Purkinje cell tumors (also known as myocardial hamartomas) were the second most common type of tumor in our patients (see Table 1). As reported by Garson and associates [6, 7], all the patients in this group were children, and all had incessant ventricular tachycardia. In 12 patients, cardiovascular collapse, characterized by hypotension or cardiac arrest, was present. One patient was asymptomatic.

All patients underwent echocardiographic studies, and then angiographic studies with electrophysiological mapping were done to determine the focus of the tachycardia. Medical therapy had failed in every patient. All patients subsequently underwent operation in which intraoperative electrophysiological mapping was followed by excision or cryoablation (or both) of the originating focus.

RHABDOMYOMA. All patients with rhabdomyomas were children (see Table 1). The most common presenting symptoms included intractable ventricular arrhythmias (4 patients) and heart murmurs (5). One of the patients with heart murmurs had accompanying cyanosis, which was caused by a pulmonary outflow tract obstruction as shown by echocardiography, and 1 had congestive heart failure. Three patients were known to have tuberous sclerosis, which has been noted in up to 50% of patients with rhabdomyomas [8]. Five patients had single tumors, and 4 had multiple tumors.

After attempts to treat the arrhythmias with medication failed, those patients underwent cardiac catheterization and electrophysiological mapping to delineate the arrhythmogenic foci. Two patients were found to have single tumors, and 2 had multiple tumors. They subsequently underwent operation during which intraoperative electrophysiological mapping was done, followed by excision of the tumor or tumors.

Four of the patients with heart murmurs underwent resection of the tumors. A palliative shunt was created in the patient with cyanosis because the large size of the tumor precluded resection.

FIBROMA. All patients who had a fibroma were children (see Table 1). Presenting symptoms included heart murmurs in 3 and congestive heart failure from cardiac obstruction in 2. In 1 patient, the tumor was found during evaluation for cardiomegaly and in another, during operation for pulmonary stenosis. All patients in this group had single tumors. Depending on individual conditions, operative procedures ranged from incision biopsy to complete resection.

LIPOMA. Among the 4 patients in whom lipomas were found, diagnosis was made on examination of chest roentgenograms in 2, during coronary artery bypass grafting in 1, and during aortic valve replacement in 1 (see Table 1). Despite their large sizes, these tumors caused no specific symptoms. In all patients, the tumors were completely excised.

OTHER BENIGN TUMORS. One patient had excision of a right atrial hemangioma discovered during operation to repair the mitral valve. One patient had a spherical filling defect at the mitral valve, which was caused by a cyst attached to the anterior leaflet of that valve; the patient underwent operation to remove the cyst. A 1.7-cm mass was found in the right ventricular outflow tract of a patient with a loud heart murmur. The mass proved to be a cardiac venous malformation (varix) filled with organized thrombus and was easily removed. A right ventricular angiomatous hamartoma was found in 1 patient who had premature atrial contractions associated with a lesion seen on the chest roentgenogram; the tumor was subsequently excised. One patient underwent removal of a granuloma involving the surface of the right ventricle. Finally, an intramyocardial cyst was located and removed in 1 patient.

Primary Malignant Tumors

All 12 patients had varieties of sarcoma (Table 2), and all had varying degrees of cardiac obstruction and resultant congestive heart failure. Presenting symptoms included chest pain (4 patients), weight loss (3), and arrhythmia (1 patient). Diagnosis was made through echocardiography

Table 2. Profiles of Patients With Primary Malignant Tumors

Tumor Type	No. of Patients	Sex		Age Range[a]	Deaths		Follow-up	
		M	F		Early[b]	Late[c]	No. of Patients	Duration
Angiosarcoma	4	2	2	33–64 yr (53 yr)	1	2 (6, 6)	1	17 mo
Malignant fibrous histiocytoma	2	2	0	24–35 yr (29.5 yr)	0	2 (14, 18)	. . .	. . .
Fibromyxosarcoma	1	0	1	43 yr	0	1 (2)	. . .	
Myxosarcoma	1	0	1	28 yr	1	0	. . .	. . .
Leiomyosarcoma	1	1	0	20 yr	0	1 (8)	. . .	. . .
Fibrosarcoma	1	1	0	26 yr	0	1 (9)	. . .	. . .
Undifferentiated sarcoma	2	1	1	3 mo, 10 yr (5 yr)	0	0	2	6 mo, 3.5 yr
Total	12	7	5	3 mo–64 yr (33 yr)	2	7 (9)	3	21.6 mo

[a] Data in parentheses are mean ages. [b] Early death is defined as death within the perioperative period or within 30 days of operation. [c] Data in parentheses are lengths of survival in months.

(12 patients), angiography (11), computed tomography (6), and magnetic resonance imaging (1 patient). All four chambers were involved, including the left atrium in 7 patients and the right atrium in 4. Whereas in 11 patients the disease was limited to the heart alone, 1 patient with severe symptoms of congestive heart failure was known to have hepatic and pulmonary metastases. Five patients had undergone operations at other hospitals before being referred to us for treatment. At our institution, 1 patient underwent biopsy alone, and 7 had incomplete excisions. Complete excision of the offending malignancy was attempted in 4.

Metastatic Tumors

The two most common metastatic tumors in this series were sarcomas and adenocarcinomas (Table 3). Presenting symptoms included congestive heart failure (12 patients), weight loss (8), and chest pain (5). No patient was asymptomatic. Three patients had superior vena cava syndrome. Diagnosis was confirmed by echocardiography (14 patients), angiography (12), computed tomography (8), and magnetic resonance imaging (2).

Complete excision of the tumor was attempted in 13 patients. Five patients had partial excisions, and 1 had biopsy alone. In 4 patients, including 2 of the patients with superior vena cava syndrome, the operation was performed during a brief period of hypothermic circulatory arrest. One patient with a metastatic paraganglioma involving the posterior wall of the left atrium underwent cardiac autotransplantation [9]. The heart was removed, and the posterior wall of the left atrium was resected and reconstructed with a pericardial patch.

Results

Of the 133 patients who underwent operation, 12 died early (within 30 days of operation), and of the 20 patients who died later, 15 had malignant disease. Overall opera-

Table 3. Profiles of Patients With Metastatic Cardiac Tumors

Tumor Type	No. of Patients	Sex		Age[a] (yr)	Deaths		Follow-up	
		M	F		Early[b]	Late[c]	No. of Patients	Duration
Sarcoma	6	4	2	25–53 (38.8)	2	3 (1.5, 3, 10)	1	5 mo
Adenocarcinoma	5	3	2	46–70 (59.6)	1	3 (2, 3, 3)	1	6 yr
Paraganglioma	2	1	1	40–42 (41)	1	0	1	3 yr
Melanoma	2	1	1	28–51 (39.5)	0	1 (7)	1	3 yr
Hepatoma	1	1	0	49	1	0	. . .	. . .
Renal cell carcinoma	1	1	0	49	1	0	. . .	. . .
T cell lymphoma	1	1	0	39	0	1 (4)	. . .	. . .
Hypereosinophilic syndrome	1	1	0	46	0	0	1	3.5 yr
Total	19	13	6	25–70 (46)	6	8 (4.2)	5	3.2 yr

[a] Data in parentheses are mean ages. [b] Early death is defined as death within the perioperative period or within 30 days of operation. [c] Data in parentheses are lengths of survival in months.

tive survival was 91%. Total follow-up for this series is 572.8 patient-years (mean follow-up, 5.2 years).

Benign Tumors

MYXOMA. Of the 63 myxomas, 57 (90.5%) were of left atrial origin and six (9.5%), of right atrial origin; none originated in either ventricle. The tumors ranged in size from 1.5 × 1.5 × 0.5 cm to 11 × 7 × 4 cm. The consistency of the tumors also varied from smooth and firm to friable and soft. Associated operative procedures included mitral (5 patients) or tricuspid (4) valve repair or replacement, aortocoronary bypass (3), atrial septal defect closure (2), and aortic valve replacement (1 patient).

Arrhythmias, usually transient supraventricular tachycardia, were the most common postoperative complication (12 patients). Two patients had pulmonary emboli, 1 experienced bleeding, and 1 had a stroke. Only 1 patient, who had undergone treatment for a left atrial myxoma, had a recurrence of myxoma in the right atrium after operation.

Sixty-one patients lived more than 30 days after operation, and follow-up information was available on 50 (see Table 1). For the 2 patients who died early, the cause of death was myocardial infarction on postoperative day 4 in 1 and multiorgan failure on postoperative day 28 in the other. This latter patient was 81 years old, the oldest in the series. There were three late deaths, and the only one related to myxoma occurred in a 32-year-old woman who had multiple recurrences and reoperations and died as a result of cardiomyopathy. All the other survivors are asymptomatic and free from disease.

PURKINJE CELL. Arrhythmias were successfully ablated in all 13 patients, and visible areas of gray-white myocardium were excised in 12. None had evidence of ventricular tachycardia on leaving the operating room. Three patients experienced recurrent tachycardia within the first postoperative week and required reoperation; all reoperations were successful. Postoperative complications included ventricular premature contractions in 2 patients, which resolved with antiarrhythmic medication, and complete heart block in 1 patient in whom a pacemaker was subsequently implanted. The one death occurred 3.5 months after operation in a patient with diffuse endocardial lesions that required extensive subendocardial resection and led to intractable congestive heart failure.

Twelve patients have been followed, all of whom are alive and well (see Table 1). They have neither experienced recurrent arrhythmias nor required antiarrhythmic medications.

RHABDOMYOMA. After operation, 3 of the 4 patients with arrhythmias had successful resolution of tachycardia and have not required antiarrhythmic medication. The other patient, a 4-month-old infant, died on postoperative day 1 of intractable congestive heart failure secondary to papillary muscle injury that occurred during operation. All of the patients seen with heart murmurs survived operation and were alive and well at follow-up.

FIBROMA. All patients survived operation and were alive and well at follow-up (see Table 1). One patient who was 7 months old at the time that an incision biopsy alone was performed on a left ventricular free wall tumor has been followed for 9 years and continues to do well.

LIPOMA. All 4 patients survived the operation without postoperative complications and were alive and well at follow-up (see Table 1).

OTHER BENIGN TUMORS. Each of the 6 patients survived operation, and 5 have remained asymptomatic without signs of recurrence (see Table 1). The patient with the intramyocardial cyst, however, died within two days of operation as a result of cardiac arrest. At postmortem examination, no evidence of myocardial infarction could be found.

Primary Malignant Tumors

Of the 12 patients with primary malignant tumors, 3 are alive at 6, 17, and 54 months (see Table 2). These patients all had complete excision and included both children with undifferentiated sarcoma and 1 adult with angiosarcoma. Two patients have undergone postoperative chemotherapy and 1 patient, radiotherapy.

The patients who died survived an average of 9 months postoperatively, and palliation of the obstructive symptoms was good. One patient with angiosarcoma and the patient with myxosarcoma died in the immediate perioperative period. Metastatic disease was the cause of death in all but 1 of these patients. In 2 of the patients with angiosarcoma, no evidence of residual cardiac tumor was found [10]. The 2 patients who underwent resection of primary cardiac malignant fibrous histiocytomas survived 14 months and 18 months.

Metastatic Tumor

Despite their debilitated condition, 17 of the 19 patients with cardiac metastases survived attempts at surgical resection. There were four additional early deaths, and 13 patients were discharged from the hospital with relief of symptoms. Of these, 8 died an average of 4.2 months after operation, and 5 are alive at an average follow-up of 3.2 years.

Among operative survivors, early complications included respiratory or congestive heart failure (2 patients each), arrhythmias (1 patient), and bacterial sepsis following chemotherapy (1). Except for 1 of the patients with congestive heart failure and the 1 with arrhythmias, early postoperative complications played a major role in the early deaths.

Comment

Using modern techniques of myocardial protection, cardiopulmonary bypass, hypothermic circulatory arrest, and even cardiac autotransplantation, the cardiac surgeon can resect almost all tumors of the heart and great vessels. Patients with benign tumors have a low incidence of morbidity and mortality, and resection results in cure.

As in other series [11, 12], myxomas were the most common tumors seen in our patients. Although they are considered benign because they lack mitotic aggressiveness and do not produce metastases, myxomas can cause severe physiological disruptions. Congestive heart failure resulting from intracavitary obstruction of blood flow or interference with valvular function was present in 70% of the patients in this series, and 9 required mitral or tricuspid valve repair or replacement. In addition, 27 (69%) of 39 patients had pulmonary hypertension, the degree of which seemed to correlate with the size of the tumor. Palpitations were present in 22%, and emboli, which were evenly distributed between the cerebral and peripheral blood vessels, were found in 21%. Such disruptions warrant performing surgical ablation as soon as possible after diagnosis is confirmed.

Whereas either angiography or echocardiography can be used to confirm the diagnosis, the latter method has become the primary diagnostic technique because of its high degree of accuracy and lack of invasiveness [13]. Operation can be undertaken solely on the basis of echocardiographic findings, but coronary angiography should be performed in older patients who may have coronary artery disease [11, 12].

In the surgical treatment of myxomas, we have found that using a biatrial approach allows all four cardiac chambers to be inspected, limits manipulation of the mass, and facilitates the surgeon's ability to completely excise the tumor, which decreases the possibility of local recurrence. Overall, the recurrence rate in patients with myxomas was 6.3% (4 patients), and operative mortality was 3.17%.

The next three most common benign primary tumors that we treated were Purkinje cell tumors, rhabdomyomas, and fibromas, all of which occurred in children. Although rhabdomyomas are the most common tumors seen in most other series of pediatric heart tumors [6], because of our interest in arrhythmia surgery, we saw more Purkinje cell tumors. Originally described as an autopsy finding in the pathology literature [14, 15], this lesion causes incessant ventricular tachycardia refractory to antiarrhythmic medication. Combining electrophysiological mapping with surgical excision in these patients has yielded good long-term results (average follow-up, 3.8 years) with cure of ventricular tachycardia.

Incessant ventricular tachycardia unresponsive to antiarrhythmic treatment can also indicate the presence of rhabdomyomas, although rarely [6]. There were 9 patients with rhabdomyomas, 4 of whom had incessant ventricular tachycardia, and they received identical treatment to that of the patients with Purkinje cell tumors. One patient died on postoperative day 1, whereas the others are free from arrhythmias at an average follow-up of 51 months. In addition, the 4 patients with rhabdomyoma who were seen with heart murmurs and underwent tumor resection are doing well, as is the patient in whom a palliative shunt was created. Regression of these tumors with time has been reported [16], and in the absence of arrhythmias or obstructions, it is possible to defer surgical removal. Whereas reported mortality rates have been as high as 40% in children with rhabdomyomas [17], 8 of our 9 patients continue to do well at an average follow-up of 3.5 years.

All of the children with fibromas did well after operation, with an average follow-up of 7.3 years. Although others [18] have noted that patients usually are seen with arrhythmias, heart murmurs and congestive heart failure were the most common findings in our patients. As evidenced by the case of 1 of our patients who underwent incision biopsy alone and who is alive and well 9 years later, operation can be deferred if resection is not feasible and no obstructions or arrhythmias are present. Lipomas are generally removed to obtain a tissue diagnosis, and patients normally do well after removal. We treated 6 patients for various other benign tumors, and all, except for 1 who died of unknown causes, were alive and well at follow-up.

Historically, most primary malignant cardiac tumors are sarcomas [11, 19, 20], which generally affect adults (only 2 of our patients were children). Because these tumors are highly aggressive, with local invasion and early metastases, the average time of survival is 6 months from the time of diagnosis [11]. Some investigators [12, 17] consider resection a palliative measure, the goal of which is to relieve symptoms of obstruction or tamponade, but others [21] have reported long-term survival of these patients. Whereas palliation was achieved in 7 of our patients who lived an average of 9 months postoperatively, 3 patients, 1 of whom underwent operation 3.5 years ago, are alive and free from disease at the time of writing.

Patients with metastatic tumors often are not referred for surgical treatment because the disease is usually widespread and the prognosis is poor. These patients are generally in debilitated condition, and surgical intervention is undertaken to relieve severe symptoms of pain or obstruction. In determining the need for operation, the risk of operative death must be carefully weighed against the benefits of palliation. Techniques such as hypothermic circulatory arrest and even autotransplantation can be used in resection of difficult masses. Operative survival was 68.4% in our series, and 6 patients died early. Of the 13 patients who were discharged from the hospital, all reported relief of symptoms, and some were able to return to a nearly normal life-style. Five of these patients were alive at follow-up (average follow-up, 3.2 years after operation). Patients who died late survived an average of 4.2 months.

On the basis of our 25-year experience in treating cardiac tumors, we conclude that aggressive surgical management is warranted. Cure can be achieved in patients with benign tumors, and good palliation is possible in those with malignant tumors. Life can be prolonged and cure may be possible with development of more effective adjuvant therapies.

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DISCUSSION

DR W. RANDOLPH CHITWOOD, JR (Greenville, NC): I congratulate Dr Murphy on an absolutely outstanding presentation, and Drs Cooley, Sweeney, and co-workers for what is most assuredly the largest series of patients with cardiac tumors treated surgically, that is, 133 patients. I am very supportive of your aggressive approach for the treatment of primary and metastatic malignant tumors with obstruction. Your survival of up to 4½ years for patients with primary malignant tumors and the average survival of 3.2 years for 5 patients with metastatic tumors are excellent.

I thought that the use of autotransplantation in your series was a very ingenious approach. I know of only 2 patients in the literature who had transplantation, and they had cardiac fibromas.

Certainly this is the largest group of Purkinje tumors extant, displacing rhabdomyomas in your series as the second most frequent tumor. Three patients required reoperation for recurrent arrhythmias after a Purkinje tumor resection. Also, I noticed that 1 patient died of multiple Purkinje tumors in the endocardium. At reoperation in those patients with recurrent arrhythmias, did you find multiple tumors?

DR MURPHY: The patients who had reoperation underwent a local reexcision of the Purkinje cell lesions. The mapping was done again and localized the earliest activation to the site where resection had previously been done. Resection involved a more extensive procedure, and that ultimately resulted in the death of the 1 patient who died 3.5 months after operation. She had extensive subendocardial tumor and required resection of both left and right ventricular endocardium. She died of refractory congestive heart failure.

DR CHITWOOD: The biatrial approach is certainly a very excellent method for both localization and mobilization of myxomas; however, my colleagues and I have found that the right atrial approach is satisfactory for most cardiac myxomas, including right atrial and left atrial myxomas. Even large ones can be excised through the septum. However, we do use transesophageal echocardiography very liberally as well as epicardial echocardiography for children with rhabdomyomas. Have you used transesophageal echocardiography for definitive localization of the tumor stalk?

DR MURPHY: We have not yet used intraoperative echocardiography in the evaluation of these patients with tumors. This technique appears to be becoming more popular at our institution, but is still in its earliest stages of operative use.

DR IRVING L. KRON (Charlottesville, VA): I have two questions. One, do you definitely need the biatrial approach to left atrial myxomas? Dr Nolan has demonstrated to me the technique of inverting the left atrium through the atrial appendage, and you can get excellent access to the septum.

My second question concerns Purkinje cell tumors. Consider the case of a child who has ventricular arrhythmias. Preoperative mapping is performed, and all that is demonstrated is ventricular tachycardia but no anatomical focus by echocardiography or computed tomographic scan. Do you think that alone is an indication for surgical intervention?

DR MURPHY: The first question regarded the technique of removal of myxomas. We recommend the biatrial approach because it allows minimal handling of the tumor and complete examination of both atria and both ventricles. It allows en bloc removal of the tumor with minimal handling of the tumor itself, and has proved to be the technique that we use in most instances. Your approach, though we have not used it, sounds very effective.

As for failing to localize an actual tumor preoperatively in a child with a Purkinje cell lesion and ventricular tachycardia, there is actually no current methodology that can determine without a doubt that that child has a Purkinje cell tumor before operation. Neither angiography nor echocardiography identified any of these tumors before operation in our series. We believe that a child less than 2 years old with incessant ventricular tachycardia refractory to medication deserves operative exploration on that basis alone, after preoperative echocardiography and angiography with electrophysiological study.

DR SAFUH ATTAR (Baltimore, MD): Our experience has involved 20 primary cardiac tumors and innumerable metastatic cardiac tumors. I have a few points to stress and then a few questions to ask.

Among the benign tumors, there were seven fibromas. We had 1 child who at the age of 3 years had biopsy of a fibromyxoma. Pathological study revealed definite evidence of tumor. We followed this patient for 20 years; he is currently alive and has no evidence of recurrence. What is the follow-up for the patients with fibromas in your series?

Another point is that myxomas occur mostly in the cavities of the heart rather than in the valves. The valves are mostly involved either with fibroelastomas or with malignant tumors, and malignant tumors occur mostly in children, whereas adult cardiac tumors are mostly benign. We have had 1 case of recurrence of left atrial myxoma. The septum was excised, and yet recurrence occurred three times. What should be done when there are such repeat recurrences?

Finally, in our series, the prognosis for myxomas of the right side was much worse than that for myxomas of the left, even if the tumor was resected, because of the need for removal of the valve.

My last question concerns the method for diagnosis. We have used exclusively two-dimensional echocardiography for the diagnosis. Do you think that magnetic resonance imaging would be of any additional help? Is it necessary, considering its expense?

DR MURPHY: Of myxomas?

DR ATTAR: Of all cardiac tumors.

DR MURPHY: The last question first. Echocardiography is probably the best screening device for cardiac tumors. Angiography is usually done in addition as a complementary study. As for computed tomography and magnetic resonance imaging, magnetic resonance imaging was used increasingly in the latter part of our series. It is particularly good for determining whether there is extracavitary involvement of the tumor and whether, in fact, the tumor originates in an intracavitary location or an extracavitary site.

The follow-up of the 7 children with fibromas averaged 7 years. All were doing well in follow-up, including the patient with the massive right ventricular fibroma.

Another question concerned patients with recurrent tumors. There were 4 patients with recurrent tumors in our series, 3 of whom were operated on initially at other institutions. As for operation for recurrence, we believe that wide excision of the septum with septal patch reconstruction with Dacron is the best way to prevent recurrence.

Results of Homograft Aortic Valve Replacement for Active Endocarditis

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Since July 1985, cryopreserved homograft prostheses have been used for aortic valve replacement in 10 patients, aged 2 to 77 years, with active endocarditis. Five patients had positive bacterial cultures from excised valves, and all had clinical findings of uncontrolled infection while receiving appropriate antibiotics. Homograft valves (four) or valved conduits (six) were implanted for treatment of sepsis (6 patients), congestive heart failure (3) or recurrent emboli (1 patient), and complicating native (5 patients) or prosthetic valve (5) endocarditis. Staphylococci (6 patients), streptococci (3), and *Candida* (1) were infecting organisms. Preoperatively, Doppler echocardiography showed aortic regurgitation in all patients. At operation, 9 patients had gross vegetations, 9 had single or multiple abscess cavities, and 5 had pericarditis. Complex reconstruction of the aortic valve and annulus with homograft conduits was necessary in 6 patients (3 with previous aortoventriculoplasty). Two early deaths (ventricular failure, perioperative stroke) occurred. Mean follow-up of all operative survivors was 2.1 years (range, 0.6 to 3.6 years), and one late death resulted from arrhythmia. Homograft valve regurgitation increased in 1 patient, and 7 late survivors are asymptomatic. No patient has had recurrence of endocarditis. We conclude that cryopreserved homograft aortic valve/root replacement is an effective method for management of active endocarditis complicated by annular destruction.

(Ann Thorac Surg 1990;49:619–24)

In both native and prosthetic aortic valve endocarditis, operation may be necessary before infection is eradicated by antibiotic therapy. Valve replacement during this active phase of infection is required when sepsis is uncontrolled, when congestive heart failure resulting from valve insufficiency is severe and progressive, or when vegetations embolize despite optimal medical therapy. Often, patients with active infection have extensive annular destruction and perivalvular ring abscesses that complicate insertion of aortic valve prostheses. Several operative techniques for managing such problems have been proposed [1–14]; this report reviews our recent experience with homograft valve replacement in patients with active aortic valve endocarditis.

Material and Methods

Patient Population

From June 1985 through July 1988, 80 patients had aortic valve replacement using cryopreserved homograft valves. In 10 patients, the indication for operation was active endocarditis. These patients (all male) ranged in age from 2 to 77 years (mean age, 39 years); 5 patients had infection of their native aortic valve. Each of 5 patients with prosthetic valve endocarditis had undergone multiple prior cardiac operations (mean, 2.8 prior procedures), including 3 who had prior aortoventriculoplasty (Konno-Rastan procedure) [15, 16].

Symptoms, usually spiking fevers, were observed from 11 to 150 days (mean, 26 days) preoperatively, and bacteremia was subsequently documented by multiple (mean, seven) positive blood cultures in each patient. Causative organisms were staphylococci in 6 patients, streptococci in 3 patients, and *Candida* in 1 patient. All patients received antibiotics selected on the basis of culture results before operation, and duration of drug therapy ranged from seven to 70 days (mean, 22 days).

Preoperative Doppler echocardiographic examination was performed in all patients; aortic regurgitation was graded as follows: none or trivial = 0, mild = 1, moderate = 2, and severe = 3. Preoperatively, mean grade of aortic regurgitation was 2.4. Echocardiographic-derived left ventricular ejection fraction ranged from 0.50 to 0.71 (mean, 0.63). Echocardiography showed valvular vegetations in 6 patients, and annular abscesses were identified preoperatively in 3 patients.

Three patients had varying degrees of atrioventricular conduction disturbance. One patient with previous aortoventriculoplasty had complete heart block preoperatively and had a permanent transvenous pacemaker; 2 patients had first-degree heart block. In addition, 5 patients were in congestive heart failure; 2 had angina pectoris, and 1 had recently suffered myocardial infarction caused by coronary artery embolus.

The primary indication for aortic valve replacement was persistent sepsis in 6 patients, progressive congestive heart failure in 3, and recurrent emboli in 1 patient. In 3

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Table 1. Patient Characteristics

Patient No.	Age (yr)	Previous Operations	Operative Indication	Organism	Duration of Preoperative Antibiotics (days)	Preoperative Temperature (°C)	Preop White Blood Cell Count (× 1,000/mL)
1	50	AVR (homograft), redo AVR (Hancock)	Sepsis	*Candida albicans*	12	38	6
2	77	AVR (Braunwald-Cutter), repair of periprosthetic leak	CHF	*Staphylococcus aureus*	7	39	16
3	38	AVR (Starr-Edwards), redo AVR (Starr-Edwards), redo AVR (Konno, Björk-Shiley)	Sepsis	*Staphylococcus aureus*	11	40	18
4	14	Aortic valvotomy, redo aortic valvotomy, AVR (homograft), redo AVR (Konno, St. Jude)	Sepsis	*Staphylococcus epidermidis*	70	38	11
5	41	None	Recurrent emboli	*Staphylococcus aureus*	13	39	16
6	50	None	CHF	*Streptococcus viridans*	15	38	11
7	2	Aortic valvotomy, AVR (Konno, Björk-Shiley)	Sepsis	*Streptococcus pyogenes*	24	38	19
8	12	None	Sepsis	*Streptococcus viridans*	30	37	12
9	49	None	Sepsis	*Staphylococcus aureus*	12	39	13
10	63	None	CHF	*Staphylococcus aureus*	23	37	15

AVR = aortic valve replacement; CHF = congestive heart failure.

patients, acute hemodynamic collapse precipitated emergency aortic valve replacement. Patient characteristics are summarized in Table 1.

Operative Course

Standard cardiopulmonary bypass techniques were used, and duration of perfusion ranged from 125 to 344 minutes (mean, 210 minutes). Systemic hypothermia (mean perfusate temperature, 20°C) was used in all patients, and limited periods of circulatory arrest were required in 2. Multidose cold (crystalloid or blood) cardioplegia and topical cooling were used, and the period of aortic occlusion ranged from 92 to 194 minutes (mean, 135 minutes).

Operative findings are shown in Table 2. Nine patients had valvular vegetations, 9 had annular abscesses, and 5 had pericarditis. Five patients subsequently had positive valve cultures, whereas 8 had organisms identified on Gram stain of valvular tissue or vegetations.

The variety of defects in the annulus, aorta, and ventricular septum requiring reconstruction after debridement are summarized in Table 2. Goals of repair were (1) to provide a secure anchor for valve implantation, (2) to restore aortoventricular continuity, and (3) to exclude abscess cavities from the circulation. In 4 patients, these objectives were achieved by debridement and homograft valve implantation alone. In 6 patients, a homograft conduit was required, and 3 of these patients had additional defects repaired.

Results

Perioperative Course

Two patients died early after operation; 1 patient had biventricular failure and could not be separated from cardiopulmonary bypass. Another patient suffered a fatal intracerebral hemorrhage from a mycotic aneurysm. Nonfatal complications included hemorrhage requiring reoperation in 1 patient and bacterial pneumonia in 3 patients.

Duration of hospitalization ranged from nine to 49 days (mean, 29 days), with intensive care unit stays of two to 31 days (mean, nine days). Intravenous antibiotic therapy was usually continued during the postoperative hospitalization; 2 patients were discharged on long-term oral antibiotics.

No patient had bacteremia postoperatively, and, on the average, eight blood cultures were obtained before the patient's dismissal. Early postoperative Doppler echocardiography showed only trivial or mild aortic homograft

Table 2. Operative Characteristics

Patient No.	Operative Findings	Procedure	Defect After Debridement
1	Prosthesis, multiple vegetations, large, penetrating annular abscess (NCC)	AVR with 23-mm homograft conduit, coronary ostial reimplantation	Annulus, aorta in NCC position
2	Dehisced prosthesis, multiple vegetations, annular abscesses (RCC, LCC)	AVR with 22-mm homograft valve, bypass graft to LAD, IABP	Annulus, septum in RCC, LCC position
3	Dehisced prosthesis, multiple vegetations, infected RVOT, VSD patches, purulent pericarditis, periaortic abscess	AVR with 22-mm homograft conduit, VSD closure with anterior mitral leaflet of homograft, coronary ostial reimplantation, RVOT reconstruction with bovine pericardium	VSD, RVOT, annulus and ascending aorta
4	Prosthesis, multiple vegetations, multiple annular abscesses (NCC, RCC, LCC)	AVR with 23-mm homograft conduit, coronary ostial reimplantation	Annulus and ascending aorta
5	Bicuspid aortic valve, multiple vegetations, annular abscess (RCC), fibrinous pericarditis	AVR with 20-mm homograft valve, exclusion of abscess with homograft	Annulus, septum in RCC position
6	Bicuspid aortic valve, multiple vegetations, cusp perforation, annular abscess (NCC)	AVR with 22-mm homograft valve	None
7	Dehisced prosthesis, dehisced RVOT, VSD patches, multiple vegetations, annular abscess (NCC, LCC), infected pacemaker leads, purulent pericarditis	AVR with 19-mm homograft conduit, VSD closure with side of homograft, coronary ostial reimplantation, RVOT reconstruction with bovine pericardium	VSD, RVOT, annulus and ascending aorta
8	Bicuspid aortic valve, multiple vegetations, cusp perforation	AVR with 18-mm homograft valve	None
9	Bicuspid aortic valve, multiple vegetations, annular abscesses (RCC, LCC), fibrinous pericarditis	AVR with 29-mm pulmonary homograft conduit, exclusion of abscesses with conduit, coronary ostial reimplantation, IABP, bypass graft to LAD	Septum in RCC, LCC position
10	Tricuspid aortic valve, cusp perforation, annular abscess (NCC), fibrinous pericarditis	AVR with 25-mm homograft conduit, repair of aortic defect with conduit, IABP	Annulus, aorta in NCC position

IABP = intraaortic balloon pump; LAD = left anterior descending coronary artery; LCC = left coronary cusp position; NCC = noncoronary cusp position; RCC = right coronary cusp position; RVCT = right ventricular outflow tract; VSD = ventricular septal defect.

regurgitation (mean grade, 0.6; Fig 1). At dismissal from the hospital, all patients were afebrile and had normal white blood cell counts.

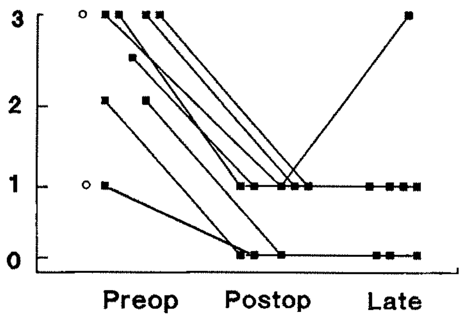

Fig 1. Severity of aortic valve regurgitation determined by Doppler echocardiography preoperatively (Preop), *postoperatively* (Postop), *and at last follow-up* (Late). *See text for description of grades of regurgitation.* Open circles *denote operative nonsurvivors.*

Late Outcome

Follow-up of operative survivors ranged from 0.6 to 3.6 years (mean, 2.1 years). One late death occurred in a patient whose sudden collapse appeared to result from arrhythmia; postmortem examination showed serious coronary atherosclerosis with no evidence of infection. Homograft valve regurgitation assessed by Doppler echocardiography increased in a single patient from mild to severe during 2.7 years of follow-up (see Fig 1). Seven late survivors are asymptomatic, and 5 of these patients are taking no cardiac medications. No patient has had evidence of recurrent endocarditis.

Comment

Aortic valve endocarditis is usually managed with antibiotic therapy initially; under ideal circumstances, valve replacement is delayed until infection is eradicated [17, 18]. When earlier operation is required, valve infection is

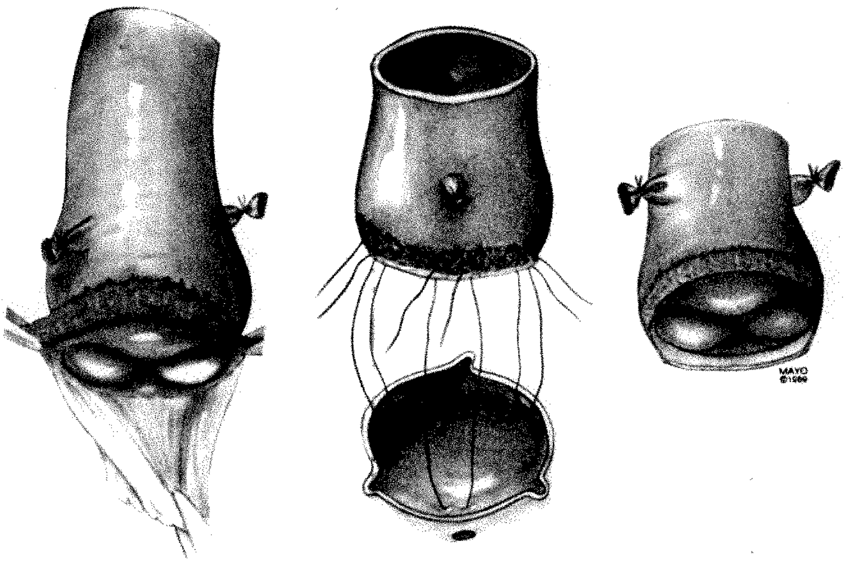

Fig 2. Complex repairs such as aortic root replacement and ventricular septal defect closure can be managed with homograft conduit implantation using the anterior mitral leaflet for septal defect closure. With annular abscesses or proximal aortic root defects, a trimmed homograft conduit may be used, with the proximal suture line in a subaortic position (mitral annulus or ventricular muscle), below the level of such defects.

frequently associated with extensive annular destruction and perivalvular ring abscesses [14, 19]. A variety of techniques for exclusion or closure of valve ring abscess cavities have been proposed; small defects can be closed by direct suture, but patch closure with Dacron or pericardium is generally preferred [10, 12, 14].

Posterior valve ring abscesses create deficiencies in aortoventricular continuity, and closure of these defects with patches is necessary to anchor the valve prosthesis securely without distorting the anterior leaflet of the mitral valve. In patients with extensive annular destruction, aortic valve translocation, conduit replacement, and extraanatomical bypass have been reported [2, 3, 6–9, 11].

Cryopreserved homograft prostheses have theoretical and practical advantages in aortic valve replacement for active endocarditis [7, 20]. First, it seems prudent to avoid use of nonbiological material in areas of bacterial or fungal contamination to minimize risk of reinfection. Data from Greenlane Hospital and others suggest that risk of early endocarditis after aortic valve replacement with homograft prostheses is substantially lower than with mechanical valves or heterografts [21, 22].

A second important advantage of homograft valves is flexibility in using the contiguous aorta and anterior mitral valve leaflet to repair abscess cavities; the valve can be individually tailored to conform to adjacent defects. Extreme examples in this series were 2 patients who had infection of aortoventriculoplasties; in these patients, all infected prosthetic material was removed and the resulting defect in the interventricular septum was reconsti-

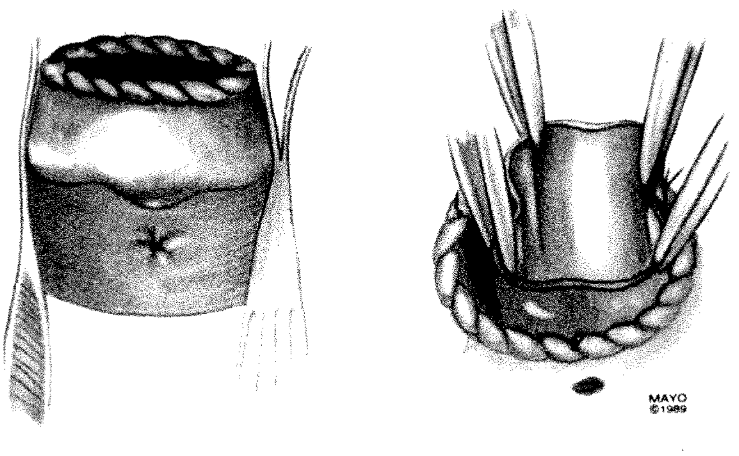

Fig 3. The proximal suture line is made with homograft conduit inverted into the ventricle. The conduit is then everted into the remnant of the native aorta.

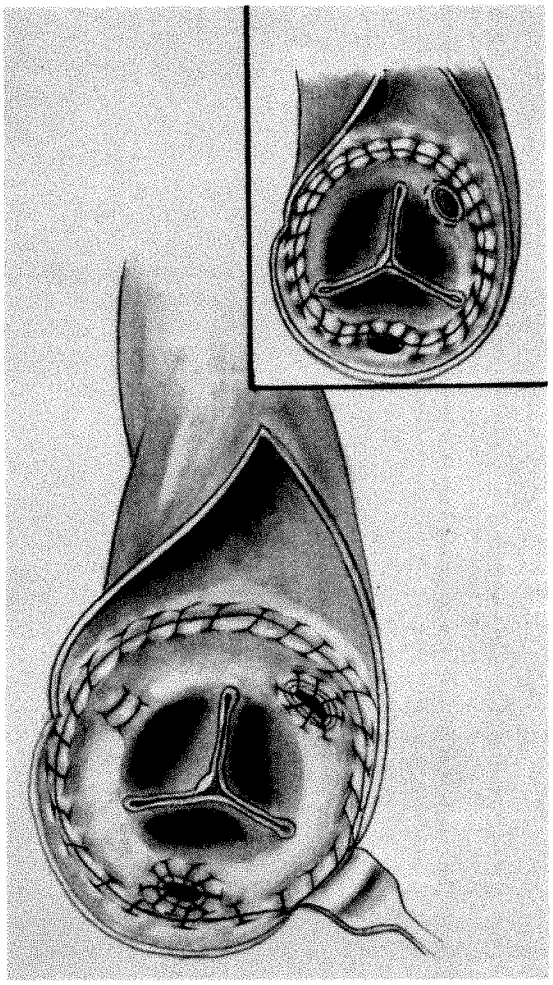

Fig 4. By placing the distal suture line above the level of any defects, the homograft may be used to reconstruct the proximal aorta and exclude annular abscesses from the circulation. Although the conduit may be scalloped to accommodate insertion of the coronary ostia (inset), we favor direct implantation of the coronary ostia into aortotomies and preservation of the sinus portion of the graft whenever possible.

tuted with the anterior leaflet of the homograft conduit or by direct suture of the muscular portion of the valve annulus to the septum.

We have used the cylinder technique for aortic valve replacement using pulmonary autografts and aortic valve homografts [23, 24]. Figures 2, 3, and 4 show the operative technique. The sinus portion of the graft is left in place, thus preserving the natural orientation and distances between the valve commissures. Coronary ostia are sewn directly to 4- to 5-mm aortotomies. We believe that this method minimizes risk of central insufficiency. The cylinder technique was extremely useful in 3 patients in this series in whom the sleeve of homograft aorta between the upper and lower suture lines excluded abscess cavities. If a properly sized aortic homograft conduit is not available, we also consider the pulmonary autograft as an aortic valve substitute; the pulmonary valve may be replaced with a pulmonary homograft conduit.

Although the present series is small, the absence of early or late reinfection encourages us to use cryopreserved homografts whenever possible for aortic valve replacement in active endocarditis. Further follow-up is necessary to determine whether late durability of homograft aortic valves will compare with that of other bioprostheses or mechanical valves.

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DISCUSSION

DR FRED H. EDWARDS (Washington, DC): Do you think it would have been possible to repair any of these valves? If not, could you comment on what role you think valve repair has in this clinical scenario?

DR TUNA: The patients with prosthetic valve endocarditis obviously required excision of their prostheses. Patients in this series with native valve endocarditis typically had extensive destruction of the leaflets and the annulus, with ring abscess formation; I do not think the valves would have been amenable in any manner to repair.

DR GEORGE R. DAICOFF (St. Petersburg, FL): These are marvelous results in a very difficult group of patients, and I think you should be congratulated. I believe that the problem with homograft valve regurgitation in the patients in this series could be solved by using the entire cuff of aorta and reimplanting the coronary arteries. I realize that this is a bigger operation and perhaps a trade-off. But the incidence of regurgitation is much higher when a freehand graft is used, as much as 30% to 35% early, and there is the worry about progressive regurgitation. You had a very low incidence of increasing regurgitation. Do you have any comments about that?

DR TUNA: We would agree with you entirely. We did have increasing regurgitation in 1 patient who had had a freehand valve scalloped in the traditional manner. That is why we have moved toward use of a homograft conduit implanted within the native aortic remnant. We believe that maintaining the sinotubular relationships and the support structures of the leaflets may prevent early and late regurgitation occurring in these valves. Selecting the appropriate size homograft in these patients can be quite difficult when the root has been destroyed and the architecture is distorted.

DR DAICOFF: Based on that experience, do you think it is worthwhile, with the additional pump time, additional ischemia, and risk of implanting the coronary arteries, of doing this as a routine aortic valve replacement rather than for infected valves only?

DR ORSZULAK: Additional time is required when replacing a standard aortic valve with a homograft. It takes longer than a conventional valve replacement. The patients presented here had extremely complex conditions. Although the bypass time was prolonged, some of these patients had to be placed on cardiopulmonary bypass before sternotomy. Thus, bypass time and cross-clamp time, because of the complexity, do not reflect the time required for standard homograft replacement. It is longer, but we believe it is justifiable in patients who are candidates for homograft. Use of a homograft may increase the required cross-clamp time by perhaps 50% over that needed for standard prosthetic valve replacement.

Controlled Initial Hyperkalemic Reperfusion After Cardiac Transplantation: Coronary Vascular Resistance and Blood Flow

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The coronary vascular response to controlled initial hyperkalemic reperfusion after global ischemia during cardiac transplantation was studied in 11 patients. The mean global ischemic time was 206 minutes (range, 143 to 245 minutes). All donor hearts received initial hyperkalemic crystalloid cardioplegia and subsequent oxygenated crystalloid cardioplegia during implantation. Coronary blood flow was highest during the first one to two minutes of controlled reperfusion but remained normal throughout the first ten minutes of reperfusion. Coronary vascular resistance was less than normal throughout the first ten minutes of controlled reperfusion, but there was a gradual increase throughout this period. Systemic vascular resistance remained within normal limits. The time to effective contraction was highly variable, but a greater potassium load during initial reperfusion was generally associated with a longer time to effective contraction.

(Ann Thorac Surg 1990;49:625–31)

As reported by the International Registry of Cardiac Transplantation, the current mortality during the first month after cardiac transplantation is approximately 10% [1], and the major cause for this mortality is acute cardiac failure after transplantation. Although many factors, such as donor management [2, 3] and alterations in recipient pulmonary vascular resistance [4], contribute to cardiac function immediately after transplantation, the major determinant of acute cardiac performance is likely the effectiveness of myocardial preservation during the three to five hours of global ischemia required for transport and reimplantation. Emery and colleagues [5] demonstrated a progressive decrease in intermediate-term survival as the ischemic time (approximated by procurement distance) increased. Profound myocardial hypothermia and cold cardioplegia have been the main components of myocardial protection during cardiac transplantation. With these techniques, ischemic periods up to approximately five hours are generally considered safe.

Experimentally and in clinical cardiac surgical studies, an initial period of reperfusion with hyperkalemic-induced asystole has been shown to provide a further increment of myocardial protection, allowing repletion of adenosine triphosphate [6–11]. The routine use of controlled hyperkalemic reperfusion in cardiac operations has been employed at our institution since 1985. These techniques were then applied to patients undergoing cardiac transplantation, and we believe this technique has contributed to more reproducibly good cardiac function immediately after transplantation.

The present study was undertaken to examine coronary vascular resistance and flow during the initial phase of controlled aortic root reperfusion after prolonged ischemia in cardiac transplantation and to provide a comparison with similar observations of the coronary vascular response to controlled reperfusion after coronary bypass grafting [12]. Such comparisons may provide insights into potential mechanisms of coronary reactivity after global myocardial ischemia.

Material and Methods

Donor Hearts and Implantation

Eleven consecutive patients undergoing primary orthotopic cardiac transplantation were formally studied. During procurement, all donor hearts received 1 L of cold (4°C) crystalloid cardioplegic solution (20 mmol KCl/L) and were immersed in cold saline solution in a plastic bag surrounded by ice slush. This maintained the myocardial septal temperature at 5° to 10°C during transport. At the time of implantation, the distal aorta was stapled shut, and a cardioplegic needle was inserted. Cold (4°C), oxygenated, crystalloid cardioplegic solution (30 mmol KCl/L) was infused initially; the infusion was repeated after 30 minutes during cardiopulmonary bypass at a systemic perfusate temperature of 25°C. Topical cooling with cold saline solution was also employed. The period of global myocardial ischemia ranged from 143 to 245 minutes (mean, 206 minutes).

Controlled aortic root reperfusion was initiated with warm blood (37°C) from the oxygenator, and the initial

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Table 1. Potassium Load of Initial Hyperkalemic Reperfusate

Volume (mL)	Potassium Concentration (mmol/L)	Potassium Load (mmol)	No. of Patients
1,000	19	19	6
500	19	9.5	4
250	19	4.75	1

phase was hyperkalemic (one to four minutes). The hyperkalemic solution was generated by drawing up blood from the oxygenator (containing approximately 4 mmol/L) and adding 15 mmol/L of potassium chloride. The total volume of hyperkalemic solution infused ranged from 250 to 1,000 mL (Table 1). This was followed by normokalemic blood (without interruption) from the pump oxygenator infused through the cardioplegic needle into the isolated aortic root with the cross-clamp still in place.

The rate of flow of the controlled aortic root reperfusion was regulated so that the aortic root pressure (continuously measured) was maintained at about 60 mm Hg (Fig 1). The aortic cross-clamp was left in place until the heart was beating vigorously with ejection into the aortic root. The distal aortic cross-clamp was gradually removed when the systolic aortic root pressure exceeded about 100 mm Hg. The left ventricle either remained collapsed (left atrial pressure less than 6 mm Hg) or was vented with a large-bore needle (13 gauge) across the interventricular septum.

Measurements and Calculations

The coronary blood flow rate was equal to the controlled aortic root reperfusion flow rate as long as the heart was asystolic, because the aorta remained cross-clamped beyond the aortic root catheter and the aortic valve was competent in each of these patients. Hemodynamic data are presented only in the interval before cardiac contraction occurred, because when a systolic pressure pulse in the aortic root was generated by cardiac systole, coronary blood flow exceeded the aortic root flow by an unknown amount.

The roller pump used to generate the controlled aortic root reperfusion flow was adjusted to be just occlusive. It was calibrated volumetrically after each operation, and a regression equation was created to convert revolutions per minute (read from a pump tachometer at closely spaced intervals during reperfusion) to flow rate in milliliters per minute. Calibrations on different days were in close agreement.

Approximately 15 minutes before the beginning of controlled reperfusion, the desired volume of blood was removed from the oxygenator reservoir to a cardioplegia reservoir (CPS-2000; Gish Biomedical Inc, Santa Ana, CA) and warmed to 37°C with a heating coil. The potassium concentration in this blood was approximately 4 mmol/L. Fifteen mmol/L of potassium chloride was added, and the blood was recirculated.

Coronary vascular resistance was calculated by dividing the aortic root pressure by the controlled aortic root reperfusion flow rate, expressed in millimeters of mercury per milliliter per minute. The coronary sinus and right atrial pressures were zero, using separate caval cannulation with loosened tourniquets to ensure a collapsed right atrium. The coronary vascular resistance and coronary blood flow were also normalized to estimated heart weight. As all of the implanted hearts were normal in size, the heart weight was estimated to be 310 grams. As a reference for normal coronary blood flow and resistance, the values for the beating heart of resting humans were used [13, 14].

Systemic blood flow was identical to arterial pump-oxygenator output during the period of controlled aortic root reperfusion. Arterial blood pressure was measured through a catheter placed in the radial artery and connected to a strain-gauge manometer. Systemic vascular resistance was calculated in the same manner as coronary vascular resistance.

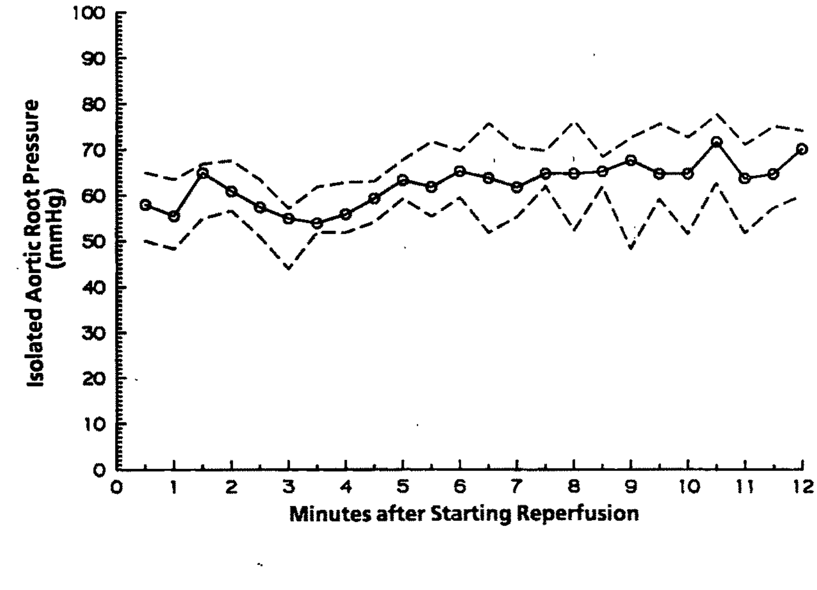


Fig 1. Mean arterial pressure in aortic root during controlled reperfusion. The circles represent the median values at each minute; the upper broken line, *the 75th percentile, and the* lower broken line, *the 25th percentile. Time zero on the horizontal axis represents the start of controlled aortic root reperfusion.*

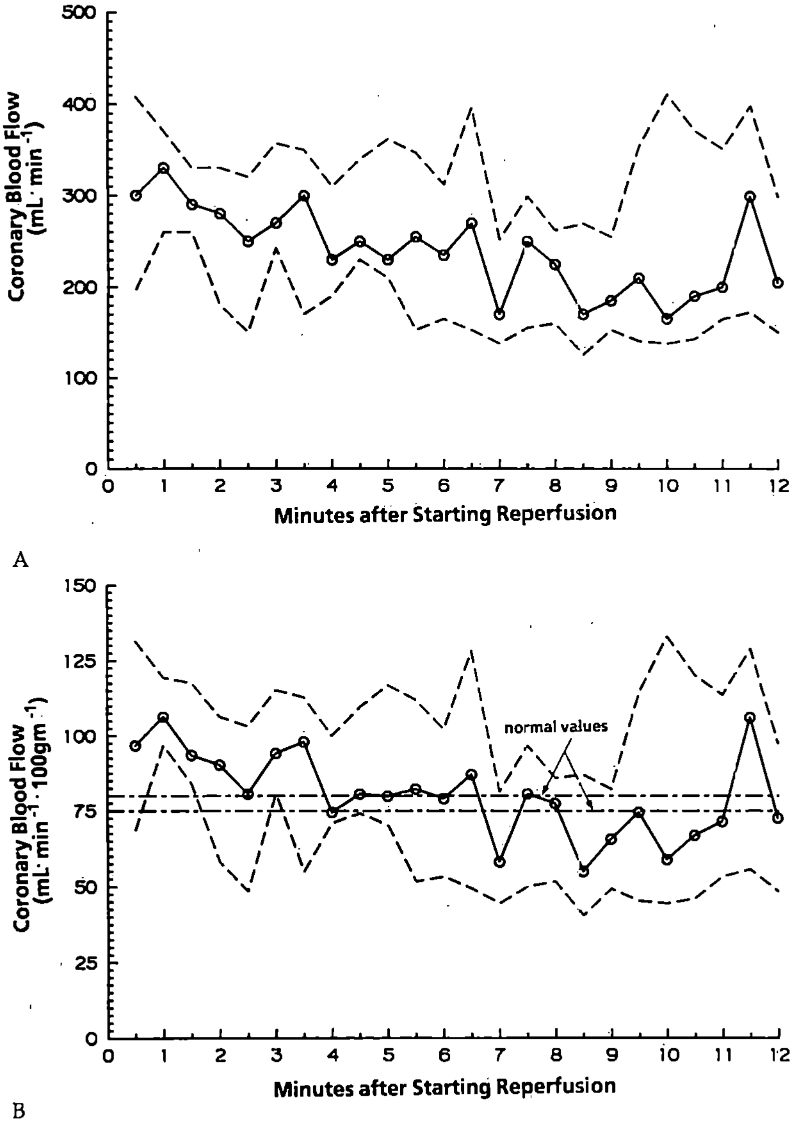


Fig 2. (A) Coronary blood flow during controlled initially hyperkalemic reperfusion (the depiction is as in Figure 1) and (B) coronary blood flow normalized to estimated heart weight. The term normal values *indicates those for the beating heart in resting humans [13, 14].*

Results

Coronary Blood Flow

There was a gradual decrease in coronary blood flow during the first ten minutes of reperfusion (Fig 2A), but these differences were likely due to chance (Table 2). When coronary flow was normalized to heart size, the values during initial controlled reperfusion were very similar to the normal values in the beating heart of resting humans (Fig 2B).

Coronary Vascular Resistance

Coronary vascular resistance increased slightly during the first ten minutes of reperfusion (Fig 3A), but the difference in resistance at one and ten minutes was likely due to chance (Table 2). Coronary vascular resistance remained at or below normal levels for the beating heart during the period of controlled asystolic reperfusion (Fig 3B).

Systemic Arterial Pressure and Resistance

With a mean systemic flow rate of 2.0 L/min/m^2 (Table 2), the mean systemic arterial pressure was approximately 60 mm Hg throughout most of the period of reperfusion (Fig 4). Systemic vascular resistance remained close to normal values during controlled aortic root reperfusion (Fig 5).

Interval Until Beating

There was considerable variability in the time of first electrical activity, with the period of asystole ranging from 2.5 to 26 minutes. The time of first beating and the time of first effective cardiac contraction (with ejection into the aortic root) were also variable; the longest interval until effective cardiac contraction was 45.5 minutes (Table 3). The time until effective contraction was directly related to the total potassium dose during controlled reperfusion, a greater potassium load resulting in longer delay until effective contraction (Table 4).

Comment

Controlled Reperfusion

Numerous experimental studies [8–11, 15–17] support the benefits of a period of initial controlled hyperkalemic asystolic reperfusion after global ischemia. Although no

Table 2. Hemodynamic Variables During Reperfusion

	Time After Starting Reperfusion (min)						
	1.0		5.0		10.0		
Variable	n	Mean ± SE	n	Mean ± SE	n	Mean ± SE	p Value[a]
Coronary flow (mL · min^{-1})	11	310 ± 25	10	290 ± 39	10	260 ± 55	0.4
Coronary flow (mL · min^{-1} · 100 g^{-1})	11	101 ± 7.9	10	95 ± 12.5	10	86 ± 17.6	0.4
Coronary resistance (mm Hg · mL^{-1} · min · 100 g)	8	0.59 ± 0.099	10	0.77 ± 0.076	10	1.1 ± 0.24	0.15
Aortic root pressure (mm Hg)	8	57 ± 4.5	10	66 ± 3.6	10	63 ± 4.8	0.4
Systemic flow (L · min^{-2} · m^{-2})	10	1.97 ± 0.046	10	2.00 ± 0.056	10	2.07 ± 0.099	0.25
Systemic resistance (mm Hg · L^{-1} · min · m^2)	9	34 ± 3.6	10	30 ± 3.0	10	30 ± 3.5	0.4

[a] p value comparing 1.0 and 10.0 minute values (paired t test).

SE = standard error.

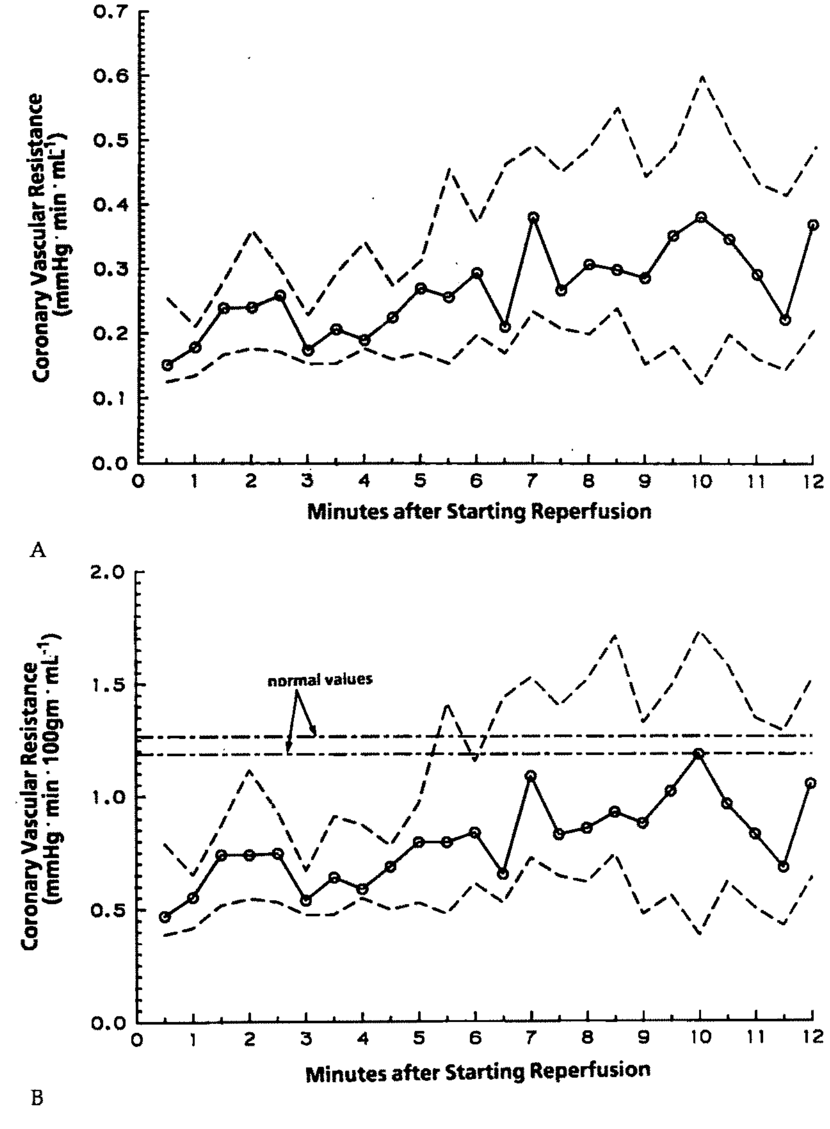


Fig 3. (A) Coronary vascular resistance during controlled aortic root reperfusion and (B) Coronary vascular resistance normalized to estimated heart weight. The depiction and definition of normal values *is as in Figure 2.*

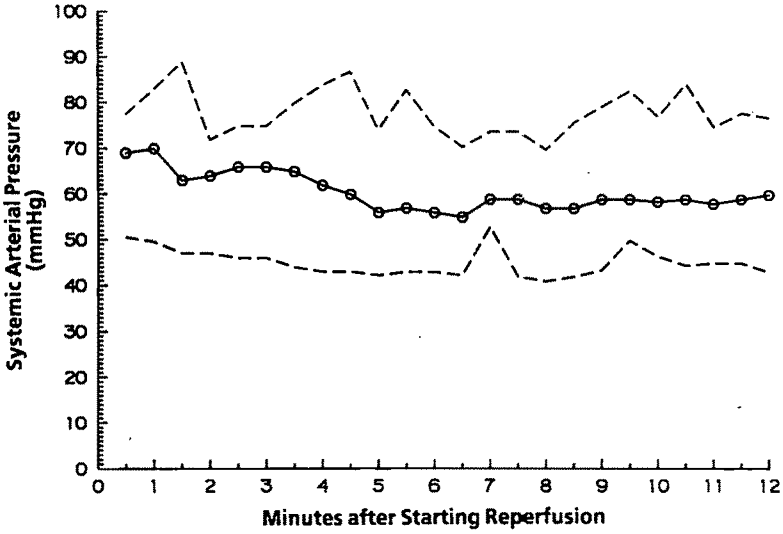


Fig 4. Systemic arterial pressure during controlled aortic root reperfusion. The depiction is as in Figure 1.

specific studies have addressed this issue in cardiac transplantation, the obligatory long ischemic periods that are often necessary underscore the potential importance of any methodology that would minimize ischemic and reperfusion injury. It is noteworthy that the average 30-day mortality after cardiac transplantation (as reported by the International Cardiac Transplant Registry) is approximately 10% [1], and the majority of these deaths are related to acute cardiac failure. In our own institution, the small but important incidence of immediate posttransplantation acute cardiac dysfunction (often isolated left ventricular failure) during the first 4 years of our experience led to the evolution of our current methodology for myocardial protection during transplantation (which includes initial cardioplegic arrest during procurement, hypothermic storage for transport at 5° to 8°C, reinfusion of two additional doses of oxygenated crystalloid or blood cardioplegia during implantation, topical cooling with cold saline solution or ice slush, and controlled aortic root reperfusion).

Coronary Blood Flow and Resistance

The low coronary vascular resistance (compared with the normal beating heart) during controlled reperfusion and the higher coronary blood flow early during reperfusion suggest an initial reactive hyperemic response after extended global ischemia [18].

The dynamics of coronary blood flow and resistance during initial reperfusion in this study differed considerably from the findings of our previous study of controlled reperfusion after coronary operations [12], in which coronary vascular resistance progressively increased (Fig 6) and coronary blood flow progressively fell during the first ten minutes of controlled reperfusion. Although the exact reasons for these differences between the transplanted normal heart and the heart with extensive atherosclerosis undergoing coronary revascularization are unknown, several possibilities exist. The endothelium of severely atherosclerotic coronary arteries is very abnormal and frequently absent, and the response to endothelial-

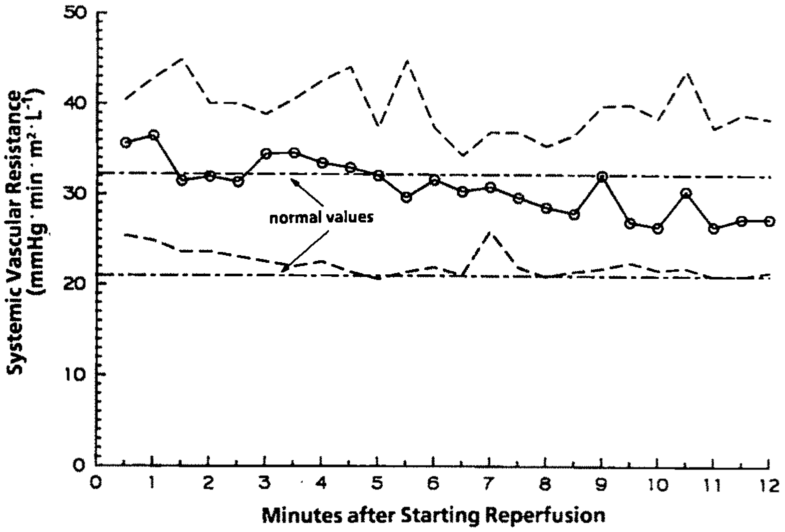


Fig 5. Systemic vascular resistance during controlled aortic root reperfusion. The depiction and definition of normal values *are as in Figure 2.*

Table 3. Onset of Cardiac Activity During Reperfusion

	Time From Beginning of Reperfusion (min)			
Event	n	Mean ± SE	Minimum	Maximum
Time of transfer hyper K+ to normo K+	11	2.5 ± 0.43	1.0	6.0
Time of first electrical activity	11	9 ± 2.6	2.5	26.0
Time of first beating (pacing)	10	16 ± 2.8	5.0	27.5
Time of first effective contraction[a]	11	26 ± 4.3	5.5	45.5
Time of first cross-clamp removal[b]	11	28 ± 3.7	10.5	49.5

[a] Effective contraction = contraction with ejection. [b] In 3 patients, the cross-clamp was removed before there was effective beating.

SE = standard error.

Table 4. Potassium Load of Initial Hyperkalemic Reperfusate

Potassium Load	Time Until Effective Beating (min)			
(mmol)[a]	n	Mean ± SE	Minimum	Maximum
4.75	1	13.5	13.5	13.5
9.5	4	24 ± 9.1	5.5	45.5
19	6	30 ± 5.2	12.0	45.5

[a] Total amount of additional potassium added to normokalemic oxygenator blood plus an assumed concentration in oxygenator blood of 4 mmol/L to produce the hyperkalemic reperfusate.

SE = standard error.

dependent relaxation factors such as acetylcholine differs markedly from normal. Werns and colleagues [19] have demonstrated a vasoconstrictive response to acetylcholine in atherosclerotic human coronary arteries, whereas normal human coronary arteries exhibit vasodilation with acetylcholine. In normal canine coronary arteries with intact endothelium (presumably the case immediately after cardiac transplantation), the administration of endothelial-dependent relaxation factors such as acetylcholine and platelet-derived adenosine diphosphate and serotonine induces vasodilation and a decrease in coronary vascular resistance [20]. Thus, the diffusely abnormal endothelium in atherosclerotic coronary arteries may contribute to the increased coronary vascular resistance during early reperfusion.

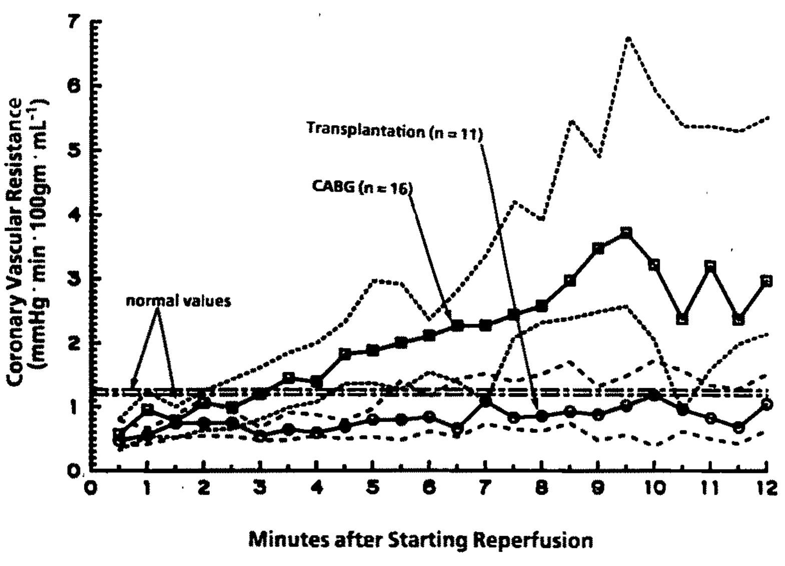
The denervated transplanted heart, with its loss of sympathetic innervation, may also have less vasoconstrictive tendency than the normal innervated heart. Finally, the variable degree of hypertrophy in the atherosclerotic heart undergoing revascularization [12] may contribute to an increased level of coronary resistance during early reperfusion as compared with the nonhypertrophied transplanted heart.

Interval Until Beating

Considerable experimental evidence suggests that a period of electrical-mechanical quiescence during initial reperfusion is advantageous to myocardial recovery after a period of global ischemia [6, 8, 9]. Rapid replenishment of adenosine triphosphate for membrane stabilization and recovery of reversibly injured myocytes is likely aided by three to five minutes of asystolic reperfusion, but prolonged periods of quiescence during reperfusion only prolong the duration of cardiopulmonary bypass without apparent additional benefit.

In this study, the time until first electrical activity ranged from 2.5 to 26 minutes with time to first effective contraction as long as 45 minutes, which is clearly excessive. This was associated with a mean cardiopulmonary bypass time of 143 minutes in these 11 study patients compared with 91 minutes for 63 consecutive patients undergoing primary or secondary cardiac transplantation between 1981 and July 1985. The direct relationship between the period of quiescence and the total potassium load during initial reperfusion suggests that a smaller total potassium load during reperfusion would provide a more

*Fig 6. Comparison of coronary vascular resistance during controlled aortic root reperfusion in patients undergoing coronary artery bypass grafting (previous published data [12]) and cardiac transplantation (*p = *0.001). The depiction and definition of* normal values *are as in Figure 2.*



desirable period of quiescence (perhaps two to six minutes) and reduce the duration of cardiopulmonary bypass.

Clinical Implications

The early (30-day) mortality reported in the International Registry of Heart Transplantation [1] as well as in our own institution between 1981 and 1985 was approximately 10%; it was largely related to acute cardiac failure immediately or early after transplantation. A major part of this mortality likely relates to inadequate preservation of the transplanted heart. Since January 1987, we have experienced only one instance (1.7%) of acute donor failure resulting in death or early retransplantation owing to acute donor dysfunction, related at least in part to the added protocols of multidose cardioplegia during implantation and to controlled reperfusion.

Our current protocol employs 250 mL of blood reperfusion containing 19 mmol/L of KCL with a total potassium load of 4.75 mmol. Despite this reduced potassium load, the duration of quiescence is more variable than that in routine coronary bypass operations.

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Atrial Pressure and Hormonal and Renal Responses to Acute Cardiac Tamponade

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The effect of acute cardiac tamponade on atrial pressures, plasma atrial natriuretic factor concentration and renin activity, and renal water and electrolyte excretion was studied in pigs loaded intravenously with hydroxyethyl starch and maintained on a continuous intravenous infusion of isotonic saline solution. Saline solution was infused into the pericardial space in 6 anesthetized pigs until a predetermined decrease of 20% in mean arterial pressure was achieved. Another 6 sham-treated pigs served as controls. Tamponade increased atrial intracavitary pressures but decreased atrial transmural (distending) pressures. These changes in atrial pressures were reversed after release of tamponade. Changes in plasma atrial natriuretic factor concentration correlated positively with changes in atrial transmural pressures. Tamponade increased plasma renin activity and decreased urine flow and renal sodium and potassium excretion, and release of tamponade reversed these changes. Thus, the tamponade-induced reduction in atrial distention is associated with hormonal changes, which may contribute to the reductions in diuresis and natriuresis observed in this connection.

(Ann Thorac Surg 1990;49:632–7)

It has been known for decades that alteration in atrial distention is associated with changes in urine flow and renal sodium excretion [1, 2]. Atrial natriuretic factor (ANF) is a recently discovered polypeptide of cardiac origin with diuretic and natriuretic properties [3]. Numerous studies have demonstrated the relationship between atrial intracavitary pressures and plasma ANF concentration [4–7]. Normally, a change in intraatrial pressure is accompanied by a parallel change in atrial wall distention. Therefore, it is difficult to separate the contributions of pressure and wall stretch in the release of ANF. Cardiac tamponade is a condition in which atrial intracavitary pressure is elevated, but transmural pressure, the true distending cavitary pressure, does not increase because of a concomitant increase in intrapericardial pressure. Therefore, cardiac tamponade offers a unique opportunity to evaluate the relative contributions of atrial intracavitary pressure and atrial transmural pressure in the regulation of ANF release. Atrial distention is also involved in renin [8] release, which may in part account for the renal responses to acute cardiac tamponade.

Recent studies in dogs [9–11] have shown no change in plasma ANF concentration during acute cardiac tamponade. Atrial transmural pressures remained unchanged [10] or decreased [11] during tamponade. The present study reassessed the relative contributions of atrial pressure and atrial distention in ANF secretion and investigated changes in plasma levels of ANF and renin during and after acute cardiac tamponade. To our knowledge, only limited and preliminary data exist regarding concomitant changes in plasma concentration of ANF and renal function in compressive pericardial diseases [12–14]. Therefore, we also evaluated the renal water and electrolyte excretion in our animals.

Material and Methods

The study protocol was approved by the Laboratory Animal Committee of Helsinki University Central Hospital. All animals received humane care in compliance with the guidelines of the "Guide for the Care and Use of Laboratory Animals" published by the National Institutes of Health (NIH publication No. 85-23, revised 1985).

Anesthesia and Preparations

Anesthesia for 12 pigs (mean weight, 14 kg; range, 10 to 20 kg) was induced with 25 mg/kg of ketamine, 10 mg of diazepam, and 40 mg/kg of azaperone intramuscularly. After induction, 6 mg/kg of sodium pentobarbital was administered intravenously, and the trachea was intubated. Anesthesia was maintained with nitrous oxide in oxygen (inspired oxygen fraction, 0.30) and enflurane (inspired concentration, 0.6% to 0.8%). After intubation, a continuous infusion of *d*-tubocurarine (0.1 mg/kg per hour) was started for muscle relaxation and prevention of shivering The animals were mechanically ventilated with a ventilator (Servo 900B) to normocarbia (arterial carbon dioxide tension, 35 to 45 mm Hg) as assessed with end-tidal CO_2 concentration and periodic arterial blood gas analyses. Surface insulation and warming blanket were used to prevent heat loss as adjusted by recording of the rectal temperature.

To induce a continuous urine flow, an intravenous infusion of isotonic saline solution (15 mL/kg per hour)

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was started after intubation and continued throughout the study. During cannulations, the animals were loaded with 30 mL/kg of hydroxyethyl starch administered intravenously within 15 to 30 minutes. The iliac artery and the iliac or jugular vein were cannulated for arterial blood pressure monitoring and blood sampling and for a vascular sheath, respectively. A 5F flow-directed thermodilution catheter with right atrial port (Edwards Laboratories, Santa Ana, CA) was advanced into the pulmonary artery. An indwelling catheter was surgically placed in the urinary bladder.

Tamponade

The pericardium was approached through a subxiphoidal skin incision. A small incision was made in the parietal pericardium, and a fluid-filled 8F catheter was advanced into the pericardial cavity directed behind the heart. The pericardial incision was closed around the catheter with a pursestring suture.

After a stabilizing period of at least 30 minutes, a 30-minute control period was started. After this control period, tamponade was induced in 6 pigs by injecting saline solution (37°C) slowly into the pericardial space through the previously placed catheter until a reduction of 20% in mean arterial pressure was observed. This was most often achieved with about 6 mL/kg of saline solution. Sixty minutes after induction of tamponade was started, the pericardial space was released by rapid gravity drainage and recovery of the fluid was confirmed. In another 6 sham-operated pigs, no tamponade was induced.

Measurements

Hemodynamic measurements included mean arterial pressure (MAP), central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), pericardial space pressure (PP), and cardiac output (CO). The pressures were measured with fluid-filled catheters connected to AE 840 pressure transducers (Aksjeselskapet Mikroelektronik, Horten, Norway) zeroed to the midthoracic level. The pressures were recorded by polygraph tracings and read at end-expiration. Cardiac output was measured in triplicate by thermodilution (model 9510 Cardiac Output Computer, Edwards Laboratories) by injecting 5 mL of saline solution (room temperature) into the right atrial port of the pulmonary artery catheter. Systemic vascular resistance (SVR) and transmural right (CVP_{tm}) and left ($PCWP_{tm}$) atrial pressure were calculated according to the following formulas:

$$SVR = \frac{MAP - CVP}{CO} \times 80,$$

$$CVP_{tm} = CVP - PP, \quad \text{and}$$

$$PCWP_{tm} = PCWP - PP.$$

Hemodynamic measurements were performed before tamponade, 30 and 60 minutes after start of tamponade, and 15, 30, and 60 minutes after release of tamponade. Urine was collected and recorded in 30-minute periods, and urinary sodium and potassium concentrations were measured from stored (at −70°C) samples.

Arterial blood samples for the determination of plasma ANF [15] and renin activity (PRA) [16] were drawn at the same stages of the study as the hemodynamic measurements were performed. Blood was taken into prechilled tubes containing ethylenediaminetetraacetic acid and 1,000 IU of aprotinin per milliliter of blood. An equal volume of saline solution was infused to replace the volume of blood sampled. The samples were centrifuged at +4°C and 3,000 rpm for ten minutes, and the plasma was stored at −70°C until assayed.

Statistical Analyses

To hold the assumptions of the analysis of variance and regression analysis, square roots of the original ANF, PRA, and renal data were taken before the analyses. Two-way analysis of variance was used to assess whether thecourses of the variables differed significantly between the groups during the entire study period. When a significant difference between the groups was noted, analysis of variance for repeated measures was applied to identify a significant change in each variable within the groups during and after tamponade. If parallel significant change occurred within both groups, the difference between the groups was tested with two-way analysis of variance. Control values before tamponade and the changes from these control values to the ending values at termination of the experiment were compared between the groups with the *t* test for independent data. Owing to repeated measures during the study, the Bonferroni method was adjusted for *p* values in the latter case. Linear regression analysis was used to assess the interrelationship between the changes from control values in cardiac filling pressures, mean arterial pressure, plasma ANF, and PRA. The null hypothesis was rejected if the probability of type I error was less than 5%. The data are given as the arithmetic or geometric (in cases of converted data) mean ± standard error of the mean.

Results

Hemodynamic and Hormonal Data

Mean arterial pressure was higher in the control group before the experiment (Table 1). All other control values of the hemodynamic and hormonal variables were similar in both groups. With the exception of systemic vascular resistance, the time courses of all hemodynamic and hormonal variables differed significantly between the groups when compared during the entire experimental period. All changes described throughout this section, ie, increases and decreases in the hemodynamic and hormonal variables induced by tamponade and release of tamponade, were statistically significant unless otherwise indicated.

EFFECTS OF TAMPONADE. Cardiac tamponade decreased transmural right and left atrial pressures, while an increase occurred in central venous pressure and pulmonary capillary wedge pressure (Fig 1). During tamponade,

Table 1. Hemodynamic Variables Before, During, and After Cardiac Tamponade in 6 Treated and 6 Control Pigs[a]

		Stage of Study					
			Tamponade (min)		Release of Tamponade (min)		
Variable	Group	Control	30	60	15	30	60
MAP (mm Hg)	T	61 ± 2	49 ± 3	47 ± 3	65 ± 2	63 ± 3	65 ± 3
	C	71 ± 2	74 ± 1	78 ± 2	76 ± 3	76 ± 4	83 ± 6
CO (L/min)	T	3.1 ± 0.4	2.3 ± 0.3	1.7 ± 0.2	3.1 ± 0.4	3.3 ± 0.4	3.0 ± 0.3
	C	3.5 ± 0.3	3.6 ± 0.4	3.6 ± 0.4	3.5 ± 0.3	3.6 ± 0.4	3.7 ± 0.4
HR (beats/min)	T	115 ± 8	116 ± 7	139 ± 11	112 ± 8	116 ± 6	115 ± 6
	C	118 ± 8	121 ± 10	124 ± 11	120 ± 9	122 ± 10	126 ± 12
SVR (dyne · s/cm^5)	T	1,465 ± 173	1,516 ± 145	1,906 ± 200	1,679 ± 185	1,458 ± 112	1,629 ± 131
	C	1,512 ± 127	1,567 ± 187	1,617 ± 166	1,630 ± 169	1,657 ± 218	1,711 ± 226

[a] Values are shown as mean ± the standard error.

C = control group; CO = cardiac output; HR = heart rate; MAP = mean arterial pressure; SVR = systemic vascular resistance; T = tamponade group.

plasma ANF concentration decreased and PRA increased (Fig 2). Mean arterial pressure and cardiac output decreased (Table 1). In the control group, no change occurred in any of these variables. Heart rate increased in both groups, but the increase was significantly greater in the tamponade group than in the control group (Table 1).

EFFECTS OF RELEASE OF TAMPONADE. As measured onward from the values at 60 minutes of tamponade, transmural right and left atrial pressures increased, while a decrease was observed in central venous pressure and pulmonary capillary wedge pressure (see Fig 1). Plasma ANF concentration increased and PRA decreased (see Fig 2). Mean arterial pressure and cardiac output increased, and heart rate decreased (Table 1). No significant changes in any of these variables were observed in the control pigs. At the end of the experiment, no difference in the changes from the control values measured before tamponade was observed between the groups in any hemodynamic or hormonal variable.

RELATIONSHIPS BETWEEN PLASMA HORMONE LEVELS, ATRIAL PRESSURES, AND BLOOD PRESSURE. When the data from both groups were pooled, the change from the control value in plasma ANF concentration was positively correlated with the corresponding changes in transmural right ($r = 0.71$, $p < 0.001$) and left ($r = 0.54$, $p < 0.001$) atrial pressures. The relationships between the change in plasma ANF and the changes in intracavitary atrial pressures were not significant. The change in PRA was not significantly related to the changes in atrial intracavitary or transmural pressures, mean arterial pressure, or plasma ANF concentration.

Renal Responses

The renal variables (Fig 3) were similar in both groups at the start of the experiment. The time courses of the renal responses to the experiment differed significantly between the groups. In the tamponade group, decreases in urine flow and urinary sodium and potassium excretion were observed during tamponade and a recovery occurred after release of tamponade. In this group, the patterns of diuresis and kaliuresis, but not that of natri-

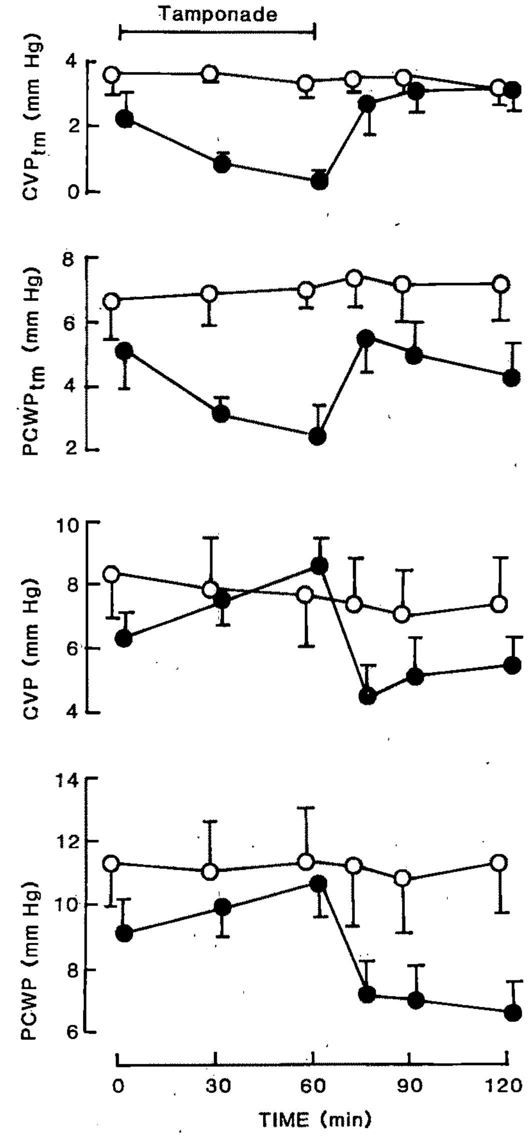

Fig 1. Atrial pressure responses to acute cardiac tamponade in 6 treated (solid circles) *and 6 control* (open circles) *pigs.* (CVP = *central venous pressure;* CVP$_{tm}$ = *transmural right atrial pressure;* PCWP = *pulmonary capillary wedge pressure;* PCWP$_{tm}$ = *transmural left atrial pressure.)*

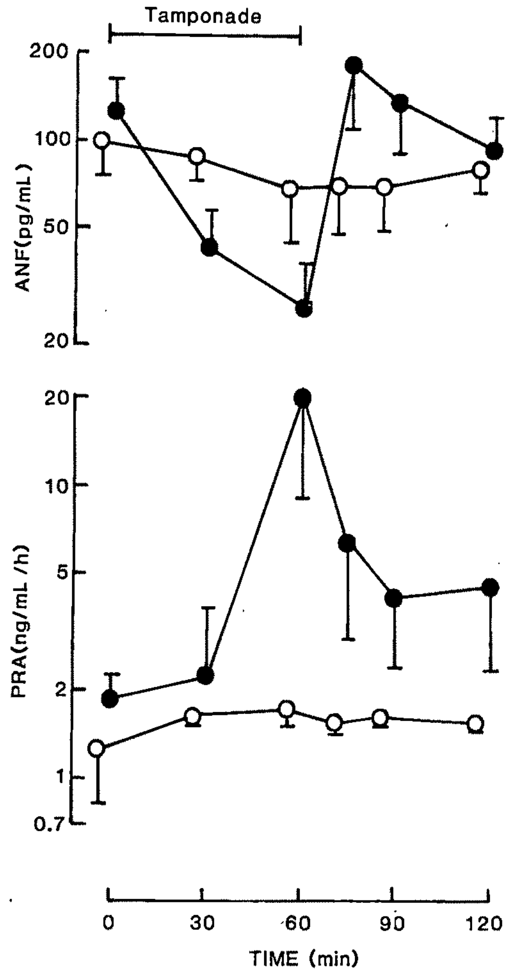

Fig 2. Plasma hormonal responses to acute cardiac tamponade in 6 treated (solid circles) *and 6 control* (open circles) *pigs.* (ANF = *atrial natriuretic factor;* PRA = *plasma renin activity.*)

uresis, were significant over time. In the control group, a time-related marked increase in diuresis, natriuresis, and kaliuresis occurred. At the end of the experiment, the changes from the control values in diuresis, natriuresis, and kaliuresis differed significantly between the groups.

Comment

By using acute cardiac tamponade as the study model, we could differentiate the individual changes occurring in distending atrial pressure and intraatrial pressure during our experiment. We found significant interrelationships between the changes in plasma ANF concentration and transmural (ie, distending) atrial pressures, but not between those in ANF concentration and intraatrial pressures during and after tamponade. Thus, our results appear to support the view that atrial stretch, not pressure, is a principal determinant controlling acute release of ANF [9, 10].

We observed a decrease in plasma ANF concentration during tamponade. In this respect, our result clearly differs from those reported previously during tamponade induced by insufflation of air [9] or instillation of fluid [10, 11] into the pericardial space in nonvolume-loaded [9] or volume-replete [10] dogs, or in dogs under conditions of fluid balance not clearly defined [11]. No change in plasma ANF level during acute cardiac tamponade was demonstrated in those studies. When the nonvolume-loaded dogs received approximately 35 mL/kg of Ringer's solution within five minutes, however, plasma ANF level was increased and then decreased for the next 15 to 30 minutes by tamponade [9]. Because the study lacked a control group, the possibility of a time-related effect must be taken into account in this decrease. Nevertheless, the normal ANF response to rapid volume loading was abolished by tamponade in these dogs, which supports our finding of decreased plasma ANF level during tamponade. We paid particular attention to the fluid balance by loading our animals with hydroxyethyl starch and maintaining them on a continuous infusion of saline solution. Therefore, our animals were probably better volume-repleted than those of other investigators. In support of this view, we measured higher plasma ANF levels before tamponade than those reported by other investigators. The volume repletion and hence higher initial plasma ANF level might have enabled us to detect the decrease in the hormone level.

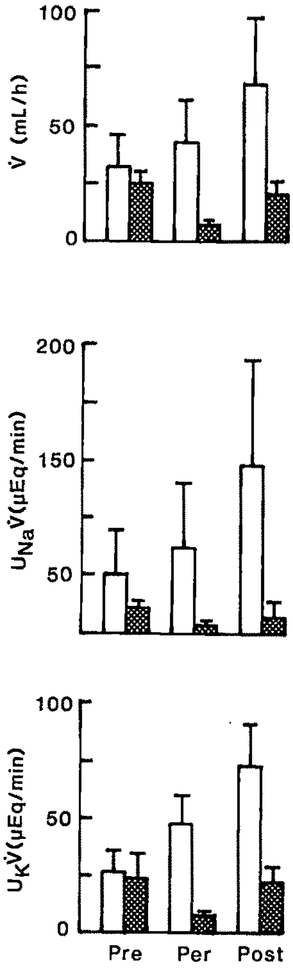

Fig 3. Renal water and electrolyte excretory responses to acute cardiac tamponade in 6 treated (hatched bars) *and 6 control* (open bars) *pigs.* (Per = *during tamponade;* Post = *after tamponade;* Pre = *before tamponade;* $U_K\dot{V}$ = *urine potassium excretion;* $U_{Na}\dot{V}$ = *urine sodium excretion;* $\dot{V}$ = *urine flow.*)

After release of tamponade, plasma ANF level increased from that measured at the end of the tamponade period in our animals. Again, the recent studies with dogs [9–11] have not been able to demonstrate any change in plasma ANF as measured five to 120 minutes after release of acute tamponade. The different management of fluid balance may explain the discrepancy in the results. In patients undergoing pericardiocentesis or pericardiotomy for cardiac tamponade [12, 13, 17] or pericardiectomy for chronic constrictive pericarditis [14], however, an increase in plasma ANF occurred after the procedures. Simultaneously, increases in atrial transmural pressures [12, 13, 17, 18] and in echocardiographic atrial volumes and atrial wall tension [13] were observed. Thus, our results and those obtained in the patient studies suggest that relief of acute or chronic constriction of the atria permits enlargement of the chambers with increased stretch and hence increased ANF release.

Increased atrial distention suppresses renin secretion [8]. In keeping with this, we found an increase in PRA during tamponade with decreased atrial distention. A similar renin response to tamponade has also been demonstrated by other investigators [9]. Activation of the renin-angiotensin-aldosterone system during tamponade has also been demonstrated by increased plasma aldosterone concentration [11]. No significant negative relationship existed between the changes in PRA and atrial transmural pressures in our animals. Therefore, further studies are needed to clarify the role of atrial stretch in the regulation of renin release during tamponade. The systemic hypotension and the decreased renal perfusion pressure induced by the tamponade and the reduced inhibition of renin release by decreased plasma ANF level [19] may also have contributed to the increase in PRA.

The hormonal changes observed during and after tamponade may have accounted for the changes in renal function in our animals. Thus, the reduction in renal water and sodium excretion during tamponade may have resulted from the decrease in ANF release and the increased secretion of renin. After release of tamponade, an inverse course in plasma levels of the hormones occurred simultaneously with a recovery in renal water and electrolyte excretion. According to two preliminary reports [12, 13] and 1 clinical case [14], relief of cardiac tamponade or constrictive pericarditis in patients can cause increased diuresis and natriuresis but only a slight increase in kaliuresis, together with an increased plasma ANF level. Similar renal changes also occur during infusion of a pharmacological dose of ANF [20]. The changes in hemodynamics should not be excluded in speculations regarding the etiological factors of renal derangement during tamponade.

Recently an acute reduction in chronically elevated intracavitary left atrial pressure was shown to result in significant reductions in urine output and sodium excretion postoperatively in patients undergoing cardiac operations for valvular or coronary artery disease [21]. The changes were accompanied by a decrease in plasma ANF level and an increase in PRA. Although the type of experimental cardiac tamponade produced in the present study may not accurately mimic that seen postoperatively, our results suggest that the major atrial determinant of hormone release in the patients [21] was the degree of distention. In any event, these findings stress the importance of atrial aspects in the renal function of cardiac surgical patients.

In conclusion, the present study of cardiac tamponade confirms previous observations that acute changes in atrial distention are accompanied by changes in renal water and electrolyte excretion. Although the hemodynamic depression produced by cardiac tamponade undoubtedly contributes to the renal derangement, the changes in plasma ANF and PRA may also have a role. The experimental model of cardiac tamponade enables us to conclude that the major determinant in the release of ANF is atrial distention rather than atrial pressure. Further studies are needed to explain the role of atrial transmural pressure in the regulation of renin release.

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Notice From the American Board of Thoracic Surgery

The American Board of Thoracic Surgery began its recertification process in 1984. Diplomates interested in participating in this examination should maintain a documented list of the operations they performed during the year prior to application for recertification. They should also keep a record of their attendance at thoracic surgical meetings, and other continuing medical education activities, for the 2 years prior to application. A minimum of 100 hours of approved CME activity is required.

In place of a cognitive examination, candidates for recertification will be required to complete both the general thoracic and cardiac portions of the SESATS III syllabus (Self-Education/Self-Assessment in Thoracic Surgery). It is not necessary for candidates to purchase SESATS III booklets prior to applying for recertification. SESATS III booklets will be forwarded to candidates after their applications have been accepted.

Diplomates whose 10-year certificates will expire in 1992 may begin the recertification process in 1990. This new certificate will be dated 10 years from the time of expiration of the original certificate. Recertification is also open to any diplomate with an unlimited certificate and will in no way affect the validity of the original certificate.

The deadline for submission of applications is July 1, 1990. A recertification brochure outlining the rules and requirements for recertification in thoracic surgery is available upon request from the American Board of Thoracic Surgery, One Rotary Center, Suite 803, Evanston, IL 60201.

Ascending Aortic Dissection in Weight Lifters With Cystic Medial Degeneration

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We report 4 cases of ascending aortic dissection in patients with long histories of weight lifting. In 2 of the patients, the initial symptoms of dissection developed while they were lifting weights. Two patients had a history of hypertension and 2 had previously used anabolic steroids. All 4 were successfully treated surgically. Histopathology showed aortic medial changes in all 4. We believe that the hemodynamic stresses of weight lifting, namely, a rapid increase in systemic arterial blood pressure without a decrease in total peripheral vascular resistance, in combination with aortic medial degeneration may have contributed to the development of the aortic dissection.

(Ann Thorac Surg 1990;49:638–42)

Several factors have been implicated in the development of aortic dissection. In patients aged more than 40 years, chronic systemic hypertension is the most common association [1]. In patients aged less than 40 years with ascending aortic dissections (Stanford type A), hypertension is less common [2, 3]. Instead, congenital abnormalities such as Marfan's syndrome, Turner's syndrome, bicuspid aortic valve, and aortic coarctation may exist [3]. Pregnancy is also a known risk factor [2].

We report 4 cases of ascending aortic dissection seen at Harbor-UCLA Medical Center between 1985 and 1989 in previously healthy young patients who were long-term weight lifters. These patients represent 50% of all cases of ascending aortic dissection seen and operated on during this period. Symptoms of dissection developed in 2 patients while they were lifting weights. Weight lifting has previously been reported to lead to subarachnoid hemorrhage [4] and brainstem ischemia [5]. A possible contributory role of weight lifting in the pathogenesis of aortic dissection is discussed.

Case Reports

Patient 1

A 22-year-old man complained in the emergency room of severe "ripping" chest pain radiating to the neck and jaw. The patient was an avid weight lifter and 6 weeks earlier, while lifting weights, had experienced a similar "ripping" chest pain that resolved spontaneously. The patient was otherwise in excellent health with no history of hypertension or previous medical problems. Vital signs were normal except for a blood pressure (BP) of 130/30 mm Hg.

Physical examination revealed a very muscular, anxious-appearing young man without a marfanoid habitus. The breath sounds were clear, and cardiac examination showed a 3/6 crescendo-decrescendo systolic murmur at the left sternal border and a 4/6 decrescendo blowing diastolic murmur at the apex. Chest roentgenogram demonstrated cardiomegaly with a normal aortic knob. Electrocardiogram was normal. An emergent aortogram showed 4+ aortic insufficiency and an ascending aortic dissection that appeared to extend into the descending aorta (Fig 1).

At operation, the ascending aorta was replaced with a 30-mm woven Dacron graft and the two commissures involved by the dissection were resuspended with horizontal pledgeted mattress sutures. The patient had an uneventful postoperative course and was discharged home on the ninth postoperative day. Histopathological examination of the aorta using the grading system of Klima and colleagues [6] showed grade 2 cystic medial change, grade 2 elastic fragmentation, grade 1 medial necrosis, and grade 3 periaortic fibrosis.

Patient 2

A 57-year-old man examined in the emergency room had a 1-week history of epigastric and substernal discomfort, progressive weakness, and shortness of breath. The symptoms had begun 1 week earlier while he was lifting weights. He was otherwise in excellent health, with no history of hypertension or other medical problems, and lifted weights seven days a week. Vital signs were normal, including a BP of 120/48 mm Hg in both arms.

Physical examination revealed an anxious-appearing, muscular male without a marfanoid habitus who was in mild distress. Bibasilar rales were present. Cardiac examination showed a grade 2/6 early systolic murmur and a grade 3/6 harsh diastolic murmur at the left lower sternal border. Chest roentgenogram showed cardiomegaly with bilateral pulmonary edema. The electrocardiogram was normal. An emergent aortogram showed severe aortic

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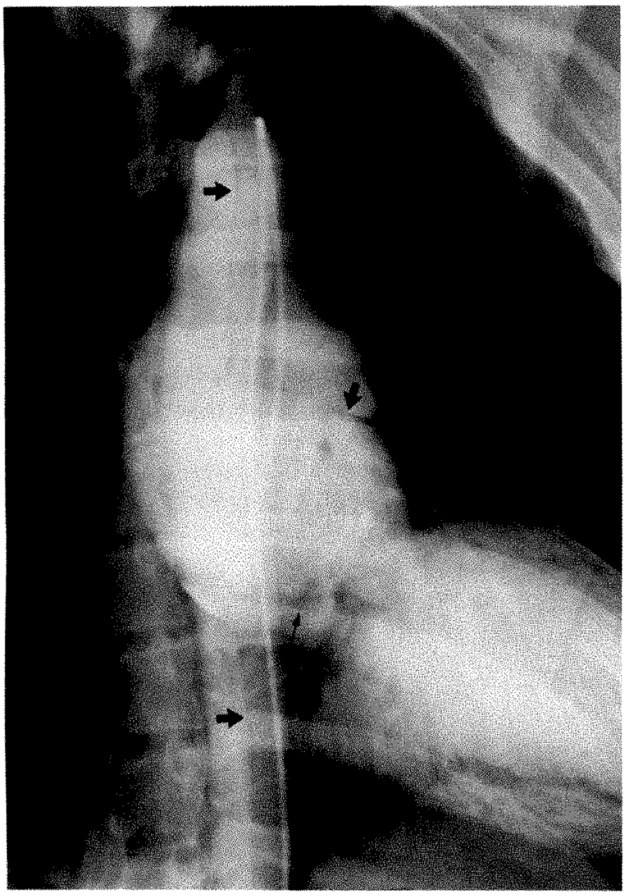

Fig 1. Aortogram of patient 1 shows a dilated aortic root with gross regurgitation into the left ventricle. Bold arrows *indicate the intimal flap.* Thin arrow *shows the proximal extent of the dissection.*

regurgitation and an intimal flap in the aortic root with a false lumen extending to the transverse aortic arch (Fig 2).

At operation, a near-circumferential tear was noted in the midascending aorta. The false lumen extended to the base of the innominate artery. The aortic valve, which was judged to be nonreparable, was excised, and a preclotted 29-mm Björk-Shiley aortic valve graft conduit was inserted. The coronary arteries were revascularized using two reverse saphenous vein grafts from the innominate artery because the normal anatomical position of the coronary ostia prevented their direct anastomosis to the graft. The patient's postoperative course was uneventful, and he was discharged home on the tenth postoperative day. Histopathological examination of the aorta showed grade 2 cystic medial change, grade 2 elastic fragmentation, and grade 1 medial necrosis.

Patient 3

A 34-year-old man was examined in the emergency room after a brief syncopal episode associated with shortness of breath, diaphoresis, and intermittent burning substernal chest pain during the previous week. He had a history of mild hypertension controlled with hydrochlorthiazide. He was a national-class weight lifter and had lifted weights for many years. He admitted to having used anabolic steroids 8 years earlier for 2 years. Vital signs included a temperature of 36.7°C, a heart rate of 118 beats/min, BP of 148/68 mm Hg, and a respiratory rate of 34/min.

Physical examination showed an anxious-appearing man without a marfanoid habitus who was in mild respiratory distress. He was muscular and weighed 122.4 kg. Bilateral rales and ronchi were present. Heart examination showed a grade 3/6 systolic murmur and a grade 3/6 diastolic murmur at the left sternal border. Chest roentgenogram demonstrated a widened mediastinum and an epicardial fat pad sign consistent with a pericardial effusion. Electrocardiogram demonstrated left ventricular hypertrophy. Laboratory values included a white blood cell count of 10,600/μL and a hematocrit of 0.45. An aortogram showed aortic root dilatation and aortic regurgitation, but no evidence of aortic dissection.

During this time, the patient's temperature and white blood cell count increased to 38.3°C and 16,300/μL, respectively. He was given intravenous antibiotics. An echocardiogram showed a large pericardial effusion, and pericardiocentesis yielded 1,000 mL of bloody fluid with a hematocrit of 0.47. Because of a high index of suspicion for aortic dissection despite a negative aortogram, cardiac cineangiography was performed and showed an intimal flap in the ascending aorta that was confirmed at operation.

As the aortic valve was not suitable for resuspension, the repair was performed using a 27-mm St. Jude valve graft conduit. The coronary arteries were bypassed using two reverse saphenous vein grafts because the normal

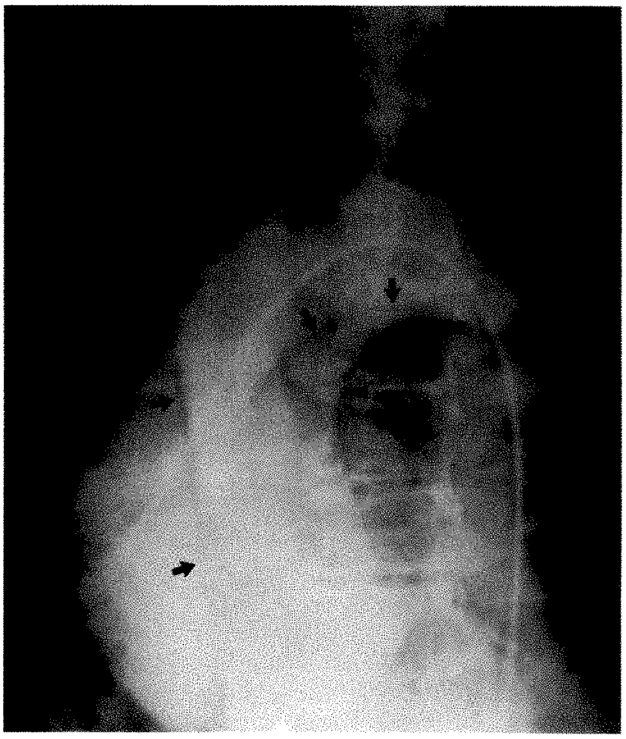

Fig 2. Aortogram of patient 2 shows a markedly dilated ascending aorta. Arrows *show the intimal flap extending from the valve to the transverse portion of the aortic arch.*

anatomical position of the coronary ostia precluded their direct anastomosis to the graft. Blood and pericardial fluid cultures were negative. The patient's postoperative course was uneventful, and he was discharged home on the ninth postoperative day. Histopathology of the aortic wall showed grade 3 cystic medial change, grade 3 elastic fragmentation, grade 3 medial necrosis (Fig 3), and grade 3 periaortic fibrosis.

Patient 4

A 37-year-old man was examined at an outside hospital because of excruciating substernal chest pain that developed while he was straining at stool. He was an avid weight lifter who had lifted weights three times a week for 15 years and had just completed a rigorous weight-lifting session. The patient had a history of hypertension controlled with clonidine and had previously abused anabolic steroids, cocaine, and heroin. Cardiac catheterization at another institution showed an ascending aortic dissection. The patient refused operation, however, and left the hospital against medical advice.

The patient came to our emergency room 5 months later complaining of dyspnea on exertion and mild substernal chest pain. Vital signs were normal and included a BP of 138/86 mm Hg. Physical examination showed a muscular man without a marfanoid habitus and in moderate distress. Breath sounds were clear, and cardiac examination showed a grade 2/6 systolic murmur and a grade 2/6 blowing diastolic murmur at the left sternal border. Chest roentgenogram demonstrated cardiomegaly but no mediastinal widening. Electrocardiogram was consistent with left ventricular hypertrophy. Echocardiography showed a dilated aortic root with a visible intimal flap and a large pericardial effusion. Cardiac catheterization confirmed the presence of an ascending aortic dissection and demonstrated severe aortic regurgitation as well as a large pericardial effusion. Pericardiocentesis yielded 1,800 mL of serosanguinous fluid.

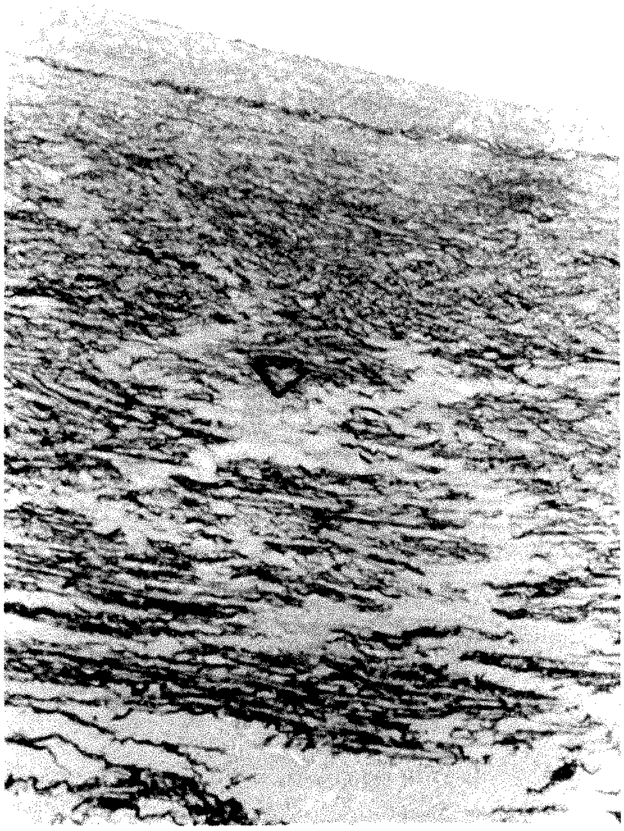

Fig 3. Histopathological section of ascending aorta (patient 3) shows medial necrosis (arrow) *as well as elastic fragmentation of focal cystic medial change (elastic stain,* ×*250 before 29% reduction).*

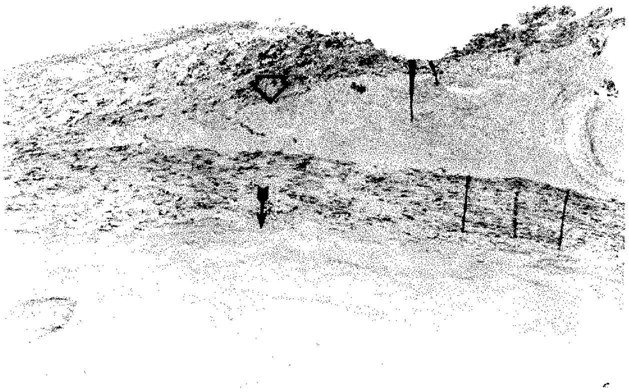

Fig 4. Histopathological section of ascending aorta (patient 4) shows complete separation of the inner and outer layers of the aortic media (clear arrow)*, extensive elastic fragmentation in the media, medial necrosis, and extensive periaortic fibrosis* (solid arrow) *(elastic stain,* ×*100 before 50% reduction).*

At operation, an intimal tear was found in the proximal ascending aorta. The prolapsing aortic valve commissure was resuspended with pledgeted horizontal mattress sutures, and the aorta was reconstructed with a 30-mm low-porosity preclotted intraluminal graft. The postoperative course was complicated by the development of a left lower lobe pneumonia, which resolved with intravenous antibiotics. The patient was discharged home on the tenth postoperative day. Histopathological examination of the aorta showed grade 1 cystic medial change, grade 3 elastic fragmentation, grade 2 medial necrosis, and grade 3 periaortic fibrosis (Fig 4).

Comment

The pathogenic mechanism of aortic dissection is not entirely defined. The most likely explanation is that an intimal tear occurs secondary to the hemodynamic forces applied to the aorta. Chronic systemic hypertension accentuates these forces and promotes the formation of atherosclerotic plaques, which may be a source of intimal tears. Hypertension also weakens the media, which may lead to aneurysmal dilatation [7]. Aneurysmal dilatation, by the law of Laplace, leads to a great increase in wall tension [8], further stressing the aorta. Once an intimal tear occurs, propagation of the dissection through the media is facilitated by an elevated BP, a steep pulse wave, and disease in the aortic media [9]. In one large series, hypertension was present in 406 of 614 patients (74%) [1].

Medial disease is found in most pathological specimens with aortic dissection. Klima and colleagues [6] histologically examined 339 aortic specimens from patients with ascending aortic aneurysms and devised a grading system modified from those of Carlson and Schlatmann. Histopathological abnormalities were divided into cystic medial change, elastic fragmentation, medial necrosis, atherosclerosis, periaortic fibrosis, periaortic inflammation, and thickening of the vasa vasorum. Using this grading system, Klima and colleagues [6] showed that the most common morphological change was elastic fragmentation in 94%, followed by cystic medial change and medial fibrosis. With this grading system, clinically significant medial changes were found in all 4 patients in our series.

Cystic medial change and medionecrosis were once thought to be a requisite for aortic dissection. Histopathological studies on nondissected aortas indicate, however, that medial degeneration occurs as part of the aging process [10]. In one study, cystic medial necrosis was found in only 13% of patients aged less than 40 years but was present in 50% of patients aged more than 40 years [11]. In the same study, hypertension was more common in patients with cystic medial necrosis. These findings have led some investigators to conclude that medial degeneration is the result of aging and is accentuated in patients with hypertension [12]. In our series, all 4 patients had some degree of medial disease and 2 had a history of hypertension, but only 1 was aged more than 40 years.

Several reports indicate that predisposing conditions leading to dissection are different in type A (those originating in the ascending aorta) versus type B (those originating in the descending aorta distal to the left subclavian artery) aortic dissections. First, patients with type A dissections tend to be younger [13]. In one series of 108 patients, the average age of patients with type A dissections was 49 years in contrast to an average age of 60 years for type B [14]. In another series, 32 patients aged less than 40 years with aortic dissection were analyzed; 87% had type A. Second, patients with type A dissections are more likely to have congenital defects predisposing to aortic dissection. In Gore's series [3] of patients aged less than 40 years, 44% had congenital defects known to predispose to dissection, such as bicuspid aortic valve, Marfan's syndrome, and coarctation of the aorta. Third, type A patients are less likely to have a history of systemic hypertension [14–16]. In the series of Applebaum and co-workers [14], only 30% of patients with type A dissection were hypertensive whereas 71% with type B were hypertensive, and, if the subgroup of type A patients aged less than 40 years is considered, hypertension is even less likely [2, 3]. In our series, all 4 patients had a type A dissection, 3 were aged less than 40 years, 2 had a history of hypertension, and none had any congenital defects.

The influence of anabolic steroids and cocaine on the development of ascending aortic dissection is unclear. Anabolic steroids are known to increase levels of low-density lipoprotein cholesterol and decrease levels of high-density lipoprotein cholesterol [17]. Acute myocardial infarction was recently reported in a 22-year-old world-class weight lifter using anabolic steroids [17]. Our 2 patients who had previously used steroids had discontinued their use several years earlier and had no evidence of serious atherosclerosis in their excised aortic specimens. Cocaine increases systemic BP markedly and has been described in association with aortic dissection and aortic rupture [18, 19]. Our 1 patient who abused cocaine, however, was not using cocaine at the time his symptoms developed.

Physical exertion or strain has previously been reported to precede aortic dissection. Hirst and associates [20] reported that of 167 patients 13% reported strenuous physical exertion before the onset of dissection. Hirst and associates [20] did not believe that the physical activity played a major role in the dissection process. Although straining or exertion cannot be implicated directly in the development of dissection, our series is remarkable in that all 4 patients had a long history of rigorous weight lifting (including 1 national-class lifter) and that symptoms developed in 2 while they were lifting weights.

Several studies have analyzed the hemodynamic effects of weight lifting. MacDougall and colleagues [21] reported that lifting heavy weights resulted in an increase up to fourfold in both systolic and diastolic BP. The increase in BP resulted primarily from an increase in cardiac output, with a lesser contribution from reflex vasoconstriction in the vascular beds of the nonexercising muscles. The increased cardiac output was thought to be secondary to an increased heart rate combined with an increase in stroke volume. The Valsalva maneuver during static exercise further increases intrathoracic pressure and may further increase BP. Another study analyzed the BP response to static contraction of different size muscle groups and found that the larger the muscle group involved in the static exercise, the greater the increase in BP and heart rate [22]. Bezuchua and co-workers [23] compared the hemodynamic responses of static (quadricep in leg extension) and dynamic (two-leg cycling) exercise. Although BP increased in both exercises, the total peripheral resistance actually increased slightly during static exercise, whereas it decreased by 60% with dynamic exercise.

Therefore, weight lifting may have contributed to the development of aortic dissection in our patients, particularly as these individuals lifted weights regularly for many years. Weight lifting clearly leads to a great transient increase in BP which, over a period of years, as might be anticipated in bodybuilders and powerlifters, may have placed critical hemodynamic stresses on the aorta. Because all our patients showed evidence of medial degeneration, the question that must be answered is whether the episodic hypertension created during weight lifting in these individuals led to their medial degeneration or if their preexisting medial disease predisposed them to aortic dissection. It seems reasonable to conclude, however, that once an aortic intimal tear occurs in the presence of both elevated BP and preexisting aortic medial disease the conditions are right for propagation of an aortic dissection. Therefore, although we cannot state unequivocally that weight lifting produces aortic dissection, we strongly believe that individuals who have evidence of cystic medial disease or family histories of this disease should avoid weight lifting. Studies investigating the long-term effects of chronic weight lifting must be performed to investigate further the possibility that chronic weight lifting and its obligatory intermittent markedly elevated BP may produce aortic wall changes resulting in acute dissection.

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Determinants of Survival and Valve Failure After Mitral Valve Replacement

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A prospective evaluation of 333 consecutive patients undergoing isolated mitral valve replacement between 1982 and 1985 was performed to identify the predictors of survival and valve failure. Follow-up between 2 and 6 years postoperatively (mean, 32 ± 17 months) was 98% complete. Four prostheses were inserted to permit a prospective evaluation of alternative valves: Björk-Shiley mechanical (n = 118), Ionescu-Shiley pericardial (n = 146), Carpentier-Edwards porcine (n = 38), and Hancock pericardial (n = 31). Hospital mortality was 6%, and actuarial survival at 5 years was 74% ± 5%. Multivariate Cox regression analysis identified advancing age (<40 years, 88% ± 7%; >70 years, 50% ± 14%) and poor left ventricular function (ejection fraction <0.20, 62% ± 17%; ejection fraction >0.60, 80% ± 7%) as independent predictors of postoperative survival. Freedom from structural valve dysfunction, prosthetic valve endocarditis, reoperation, and valve-related mortality and morbidity were 86% ± 4%, 91% ± 4%, 81% ± 4%, and 72% ± 5%, respectively, at 5 years. The actuarial incidence of valve failure was inordinately high with the Hancock pericardial valve ($p < 0.05$). Freedom from thromboembolic events (78% ± 8% at 5 years) was significantly lower in patients with poor ventricular function (ejection fraction (<0.20, 54% ± 20%; ejection fraction >0.60, 73% ± 11%; $p < 0.05$). Survival after mitral valve replacement was determined by age and left ventricular function. Premature failure of the Hancock pericardial valve resulted in an unacceptable rate of valve-related complications.

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Although the results of mitral valve replacement have improved, the mortality and morbidity of mitral valve operations remains considerable [1–5]. Identification of current risk factors may assist in the development of new strategies to improve the results in high-risk patients undergoing mitral valve replacement. In this study, a variety of prostheses were used to allow a prospective evaluation of their performance. The influence of the prosthesis on patient mortality and morbidity was examined.

Material and Methods

Patient Population

Three hundred thirty-three patients who underwent mitral valve replacement between January 1982 and December 1985 were prospectively evaluated. Patients undergoing concomitant coronary artery bypass (n = 65) or tricuspid annuloplasty (n = 31) were included in the study. During this period, 114 patients underwent combined aortic and mitral valve operations and 15 patients underwent aortic, mitral, and tricuspid valve operations and were not included in this analysis. In addition, 134 patients underwent mitral valve repair and were excluded. Younger patients who had pure mitral stenosis usually on a rheumatic basis who did not have severe valvular calcification and who did not have a history of thromboemboli underwent mitral valve repair including a mitral commisurotomy with removal of fibrotic material from both the leaflets and the subvalvular apparatus. Patients who had pure mitral insufficiency usually on a basis of myxomatous degeneration usually localized to the posterior leaflet underwent mitral valve repair rather than replacement. The incidence of mitral valve repair has increased in recent years as our capability to correct the valvular dysfunction has improved. Preoperative, operative, and postoperative data were collected prospectively.

Clinical Presentation

Sex, age, preoperative New York Heart Association (NYHA) functional class, preoperative endocarditis, urgent operations, and history of previous sternotomy were recorded prospectively. Most patients were women (64%), and 39% were aged more than 60 years. The majority of patients were in NYHA class III (63%) or IV (20%). Twenty-three patients (7%) underwent mitral valve replacement for mitral valve endocarditis. Thirty-seven patients (11%) underwent urgent operations (within 72 hours of cardiac catherization) because of ischemic mitral regurgitation (n = 17), infective endocarditis (n = 11), or prosthetic valve dysfunction (n = 9). Ninety-eight patients (29%) had undergone previous valvular operations includidng open (16) or closed (56) mitral commissurotomies, and 26 patients had previous valve replacement.

Cardiac Catheterization

Cardiac catheterization data were reviewed to determine the valvular lesion, ventricular function, and extent of the

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Table 1. Cardiac Catheterization Data

Variable	n (%)
Mitral valve lesion	
Stenosis	104 (31)
Regurgitation	134 (41)
Mixed	92 (28)
Coronary artery disease	
Single-vessel	30 (9)
Double-vessel	22 (7)
Triple-vessel	26 (8)
Left ventricular ejection fraction	
<0.20	10 (3)
0.20–0.40	38 (11)
0.40–0.60	59 (18)
>0.60	226 (68)

coronary artery disease (Table 1). One hundred four patients (31%) had predominant mitral valve stenosis with a mitral valve area less than 1.5 cm^2 and mitral regurgitation less than 1+. One hundred thirty-four patients (41%) had predominant mitral regurgitation with opacification of the left atrium of 2+ or greater with left ventricular contrast injection and a mean mitral valve gradient less than 5 mm Hg. Ninety-two patients (28%) had mixed mitral valve disease with findings of both mitral stenosis and regurgitation at catheterization and operation. Forty-six patients (14%) had tricuspid regurgitation, and 6 patients (2%) had tricuspid stenosis. Left ventricular ejection fraction was greater than 0.40 in the majority of patients (86%) and less than 0.40 in the remaining patients.

Coronary angiography showed a stenosis of more than 50% (diameter narrowing) of a major coronary artery supplying one of three cardiac regions (left anterior descending, circumflex, and right coronary artery) in 78 patients (23%). Thirty patients (9%) had single-vessel disease, and 22 patients (7%) had double-vessel disease. Three patients (1%) had left main coronary stenosis.

Anesthetic Management and Operative Technique

Anesthetic management and operative techniques were similar among the five surgeons. Fentanyl citrate was used for induction and maintenance of anesthesia. Moderate hemodilution (hematocrit, 0.20) and systemic hypothermia (25°C) were maintained during cardiopulmonary bypass. Multidose cold crystalloid cardioplegia was used for myocardial protection in the majority of patients (95%), and some recent patients (5%) received blood cardioplegia. Cardioplegic solution was infused every 20 minutes to maintain myocardial temperature less than 15° ± 3°C. A cooling pad provided additional topical hypothermic protection. Concomitant coronary bypass was performed with saphenous vein or internal mammary artery. Distal anastomoses were performed before valve replacement to permit vein graft infusions with cardioplegic solutions. The mean cardiopulmonary bypass time was 115 ± 42 minutes, and the mean aortic cross-clamp time was 71 ± 27 minutes.

Prosthesis

The prosthesis was selected based on the patient's age and a history of thromboembolism or bleeding disorder. Mechanical valves tended to be inserted in younger patients (mean age, 54 ± 12 years) and those with a history of thromboembolism (mechanical, 28%; bioprosthesis, 19%) requiring anticoagulation. Bioprostheses tended to be inserted in older patients (mean age, 56 ± 12 years) and those with a contraindication to anticoagulation. The surgeon's choice of prosthesis was based on discussion with the patient and his or her cardiologist. A variety of prostheses were used to permit a prospective evaluation of alternative valves: Björk-Shiley (n = 118), Ionescu-Shiley pericardial (n = 146), Carpentier-Edwards porcine (n = 38), and Hancock pericardial (n = 31). The distribution of prostheses inserted was similar among the participating surgeons. All valves are inserted with interrupted pledgetted sutures. Preservation of the papillary muscle apparatus was attempted in most patients with mitral regurgitation receiving a bioprosthesis.

Follow-up and Postoperative Events

After hospital discharge, each patient was seen in the valve clinic or contacted by telephone on a regular basis. For this report, all patients were contacted again between September 1987 and March 1988. The median follow-up period was 32 months (range, 1 to 63 months). Follow-up was 98.5% complete. Five patients were lost to follow-up.

All abnormalities encountered during follow-up were investigated. A deterioration in symptomatic recovery, a regurgitant murmur, or any other indication of valve dysfunction was investigated by Doppler echocardiography, cardiac catheterization, or both. Patients with deterioration of symptoms postoperatively were carefully interrogated, and a postoperative NYHA functional class was determined. The following postoperative events were considered: death, structural valve dysfunction, nonstructural valve dysfunction, prosthetic valve endocarditis, thromboembolism, anticoagulant-related hemorrhage, and reoperation for valve-related complications. We used the definitions of events recommended by the "Guidelines for Reporting Morbidity and Mortality After Cardiac Valvular Operations" [6].

Statistical Analysis

Statistical analysis was performed with the SAS (Statistical Analysis System Institute, Inc, Cary, NC) and BMDP (BMDP Statistical Software, Los Angeles, CA) statistical programs. The clinical and hemodynamic data were analyzed to determine the effect of preoperative variables on postoperative survival and prosthetic valve dysfunction. Postoperative events were characterized by actuarial statistics with the Kaplan-Meier method [7]. The generalized Wilcoxon and Mantel-Cox tests were used to evaluate the effect of each predictor on postoperative events [8]. Multivariate, time-dependent, Cox stepwise regression analysis was used to specify the independent determinants of postoperative events. Step selection was based on the maximum likelihood ratio; a p value greater than 0.15 was required for removal, and a p value less than 0.10 was required for entry into the model. Results are expressed as the mean ± standard deviation in the text and as the mean ± standard error of the mean in the figures, in which the *p* values reflect the result of the univariate Cox regression analysis for the variable shown. The numbers in the figures indicate the number of patients at risk in the interval depicted.

Results

Survival

Twenty patients died perioperatively, resulting in an operative mortality of 6%. Stepwise logistic regression identified advancing age, female sex, poor ventricular function, endocarditis, and mitral regurgitation as independent predictors of operative mortality.

Thirty-eight patients died after hospital discharge, resulting in an actuarial survival of 74% ± 5% at 5 years. Valve-related complications resulted in ten deaths (prosthetic valve endocarditis, n = 4; structural valve dysfunction, n = 3; nonstructural valve dysfunction, n = 2; thromboembolism, n = 1). Cardiac events not related to the prosthesis were the cause of 12 deaths (congestive heart failure, n = 7; myocardial infarction, n = 5). The other causes of death included malignancy (n = 6), renal failure (n = 2), and hepatitis (n = 1). There were seven unexplained sudden deaths for which an autopsy was not performed. The independent predictors of survival, by Cox regression analysis, were advancing age and poor preoperative left ventricular function (Fig 1). Patients less than 40 years of age had a 5-year survival of 88% ± 5% as compared with 50% ± 14% for patients older than 70 years. Patients with a left ventricular ejection fraction greater than 0.60 had a survival of 80% ± 7% as compared with 62% ± 17% at 5 years for patients with an ejection fraction less than 0.20.

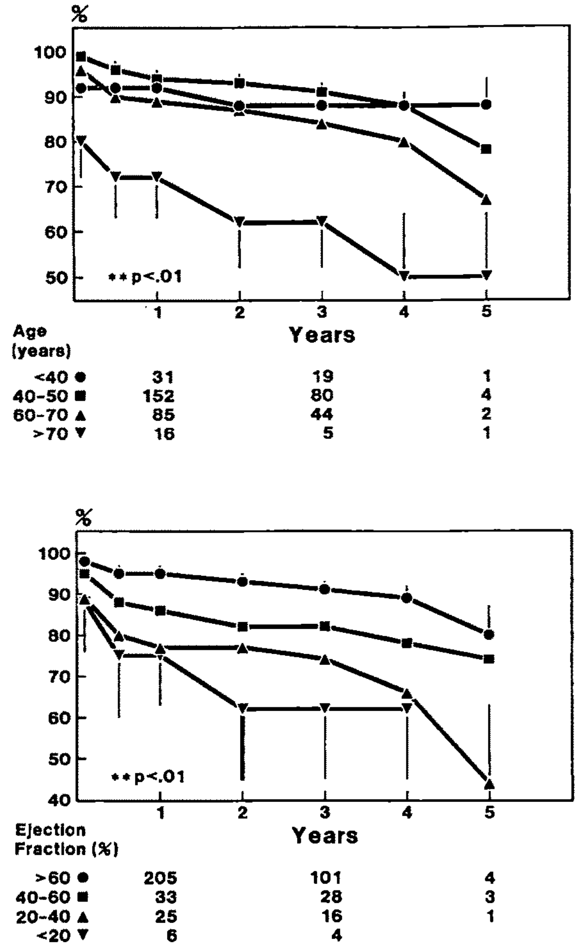

Fig 1. Overall survival was 74% ± 5% at 60 months. Survival was independently influenced by advancing age (top) and poor preoperative left ventricular function (bottom).

Postoperative Complications

Seventeen patients had structural valve dysfunction, resulting in an actuarial freedom from structural valve dysfunction of 86% ± 4% at 5 years. Structural valve dysfunction occurred in 8 patients with the Hancock pericardial valve, in 5 patients with the Ionescu-Shiley pericardial valve, and in 2 patients each with the Carpentier-Edwards porcine and the Björk-Shiley valves. Freedom from structural valve deterioration was inordinately low with the Hancock pericardial valve (Fig 2: Hancock, 22% ± 18%; Carpentier-Edwards, 86% ± 9%; Ionescu-Shiley, 95% ± 3%; and Björk-Shiley, 98% ± 2%; $p < 0.05$ by Cox regression). No other factors influencing the outcome of the Hancock pericardial valve were identified by univariate analysis, and the Hancock pericardial valve was an independent predictor by multivariate analysis.

Prosthetic valve endocarditis occurred in 12 patients, resulting in a freedom from prosthetic valve endocarditis of 91% ± 4% at 5 years. Freedom from prosthetic valve endocarditis was significantly lower in patients with previous endocarditis (80% ± 11%, n = 23, versus 92% ± 4%, n = 310) and in patients with the Hancock and pericardial prostheses (Hancock: n = 2, 82% ± 13%; Ionescu-Shiley: n = 9, 82% ± 10%; Björk-Shiley: n = 1; 98% ± 2%; Carpentier-Edwards: n = 0, 100%; Fig 3).

Twenty-nine patients underwent reoperation for valve-related complications. The indications for reoperation

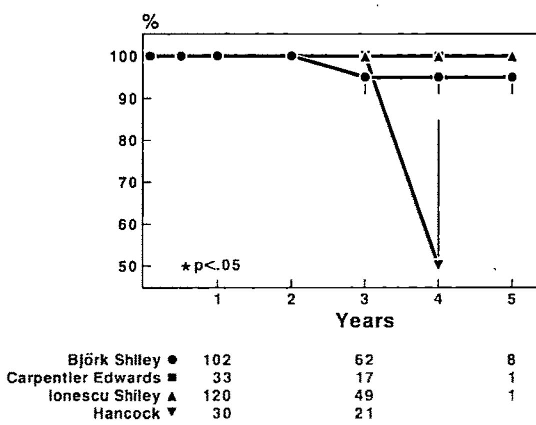

Fig 2. Structural valve dysfunction by valve type is shown. Freedom from structural valve deterioration was inordinately low for the Hancock pericardial valve.

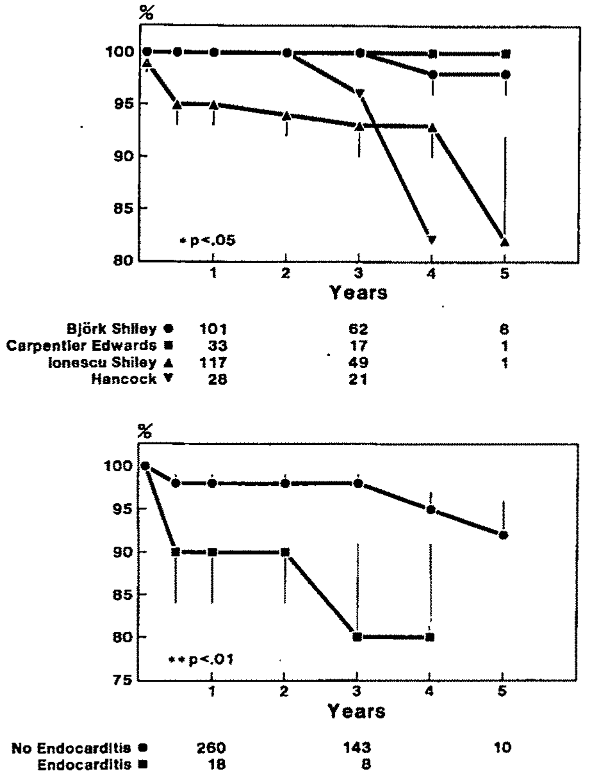

Fig 3. Freedom from prosthetic valve endocarditis was 91% ± 4% at 5 years. Prosthetic valve endocarditis occurred more frequently in patients with the Hancock pericardial valve (top) and in patients with previous endocarditis (bottom).

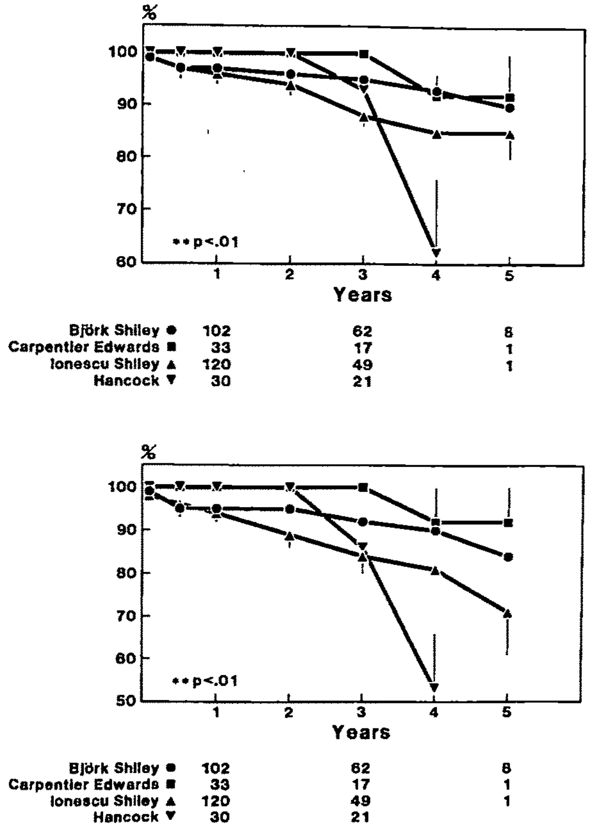

Fig 4. Freedom from reoperation (top) and valve-related mortality and morbidity (bottom) at 5 years were 81% ± 4% and 72% ± 5%, respectively. The Hancock pericardial valve adversely influenced reoperation and valve-related mortality and morbidity.

were structural valve dysfunction in 17 patients, nonstructural valve dysfunction in 6 patients, and prosthetic valve endocarditis in 6 patients. Freedom from reoperation was 81% ± 4% at 5 years and was significantly lower in patients with the Hancock prosthesis (Hancock: n = 8, 23% ± 19%; Ionescu-Shiley: n = 13, 85% ± 5%; Carpentier-Edwards: n = 1, 92% ± 8%; Björk-Shiley: n = 7, 90% ± 4%; Fig 4). Hancock valve failures also reduced the freedom from all valve-related mortality and morbidity, which was 72% ± 5% overall at 5 years (Fig 4).

Thromboembolic events occurred in 37 patients, resulting in an overall freedom from thromboembolism of 78% ± 8% at 5 years. Patients with poor ventricular function were at a higher risk of postoperative thromboembolism (ejection fraction less than 0.20, 54% ± 20%; ejection fraction greater than 0.60, 73% ± 11%; $p < 0.05$; Fig 5). The incidence of thromboembolism was not significantly different between mechanical and bioprosthetic valves (Björk-Shiley, 79% ± 9%; Hancock, 89% ± 8%; Ionescu-Shiley, 58% ± 18%; Carpentier-Edwards, 87% ± 6% at 5 years; $p > 0.05$). Thromboembolism was not influenced by the cardiac rhythm or a history of previous thromboembolic events. However, only a few patients had preoperative thromboembolic events.

The majority of patients with Björk-Shiley mitral valve replacement were anticoagulated (97%) whereas approximately one half of patients with a bioprosthesis received warfarin (Carpentier-Edwards, 54%; Ionescu-Shiley, 49%; Hancock, 38%).

Anticoagulant-related hemorrhage occurred in 20 patients with an overall freedom of 68% ± 16% at 5 years. No patient-related or valve-related factors were identified that significantly influenced this event.

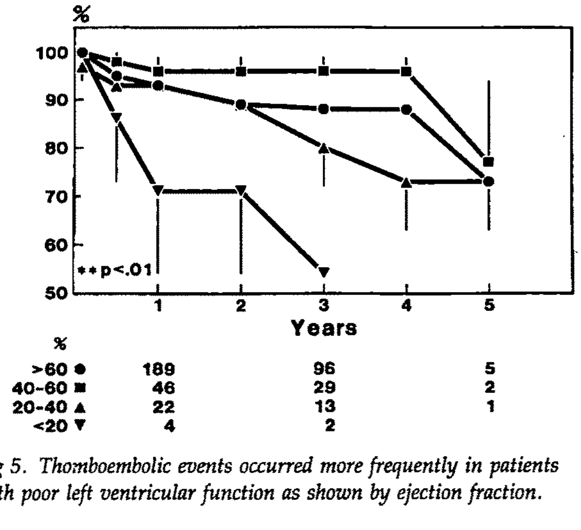

Fig 5. Thomboembolic events occurred more frequently in patients with poor left ventricular function as shown by ejection fraction.

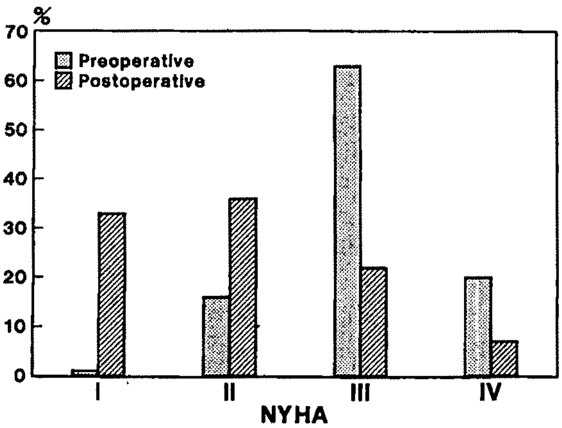

Fig 6. Preoperative and postoperative New York Heart Association (NYHA) functional class is shown. Most patients experienced symptomatic improvement after mitral valve replacement.

Postoperative New York Heart Association Class

The preoperative and postoperative NYHA functional class is shown in Figure 6. The majority of patients experienced symptomatic improvement. Sixty-nine percent of patients were in NYHA class I or II postoperatively as compared with 17% preoperatively. Ninety-two percent of patients had an improvement in one or more NYHA classes or no change if they were already in NYHA class I or II preoperatively. Seven percent of patients suffered a worsening of NYHA functional class or failure to improve from NYHA class III or IV.

Comment

This study shows the results of mitral valve replacement using contemporary surgical techniques and prostheses. Although the duration of follow-up was limited to the intermediate term, clinically significant differences were apparent.

In this report, survival was 74% ± 5% at 5 years. Advancing age and poor ventricular function adversely affected postoperative survival. Mitchell and associates [1] reported that age and the presence of congestive heart failure were significant determinants of survival. In addition, they identified valve lesion (mitral regurgitation), coronary artery disease, hypertension, and valve type as independent predictors of survival in their larger series (1,315 patients) undergoing mitral valve replacement. We did not find these factors predictive of operative mortality or postoperative survival. Neither the presence of coronary artery disease nor coronary bypass grafting influenced either operative mortality or intermediate-term survival. Cohn and associates [2] reported that survival after mitral valve replacement was affected by the valve lesion (mitral regurgitation), poor NYHA functional class, and valve type. These studies emphasized the importance of both patient and valve-related factors on outcome.

In our series, older patients and those with impaired ventricular function experienced a higher operative mortality and a lower survival at 5 years. The anticipated benefits of operation must be carefully balanced against the increased risk in such patients.

In patients with impaired ventricular function who require mitral valve replacement, improved methods of intraoperative myocardial protection may reduce operative mortality and increase longevity. Alternative techniques of myocardial protection may include use of blood cardioplegia with a warm induction [9] and a terminal warm infusion [10, 11]. Supplemental Krebs cycle intermediates [9] or calcium antagonists [12] may further improve the myocardial protection afforded by blood cardioplegia. Retrograde or coronary sinus cardioplegia may provide better delivery of cardioplegia during mitral valve replacement [13, 14]. Use of low-profile valves may permit preservation of the mitral valve apparatus in patients requiring a mechanical valve and may improve postoperative ventricular function and survival.

The Hancock pericardial valve had a high rate of failure, as reflected by the high incidence of structural valve dysfunction and the increased requirement for reoperation. Prosthetic valve endocarditis was also higher with the Hancock valve, perhaps because these abnormal valves were more susceptible to infection. Premature failure of the Hancock pericardial xenograft was recently reported by our center [15] and by others [16]. Bertolotti and associates [16] reported commissural tears in all their explanted valves and suggested that the lesions were fatigue induced [17, 18]. The findings of our study and others support this hypothesis. The Hancock pericardial valve is no longer used at our institution. Patients with the Hancock valve are being followed closely in anticipation of eventual valve dysfunction.

The Ionescu-Shiley valve was the other pericardial bioprosthesis used in these patients. Although the incidence of structural valve dysfunction was satisfactory at 5 years in this report, our follow-up at 10 years indicated a high failure rate of this valve after 5 years [19].

Postoperative thromboembolic events were significantly more frequent in patients with poor ventricular function as reflected by ejection fraction. Hetzer and associates [20] identified atrial fibrillation, history of systemic emboli, the presence of atrial clots, and low output state as predisposing factors for thromboembolic events after mitral valve replacement. Mitchell and associates [1] reported that congestive heart failure (likely as a result of impaired ventricular function) increased the likelihood of thromboembolism. Anticoagulation of patients with poor ventricular function and low output states after mitral valve replacement may reduce the incidence of postoperative thromboembolism.

The incidence of thromboembolism was not different between mechanical and bioprosthetic valves despite our preference for inserting a mechanical valve in patients with a history of thromboembolism. The higher risk of thromboembolism in these patients with mechanical valves was probably reduced by postoperative anticoagulation.

In summary, survival after mitral valve replacement was determined by age and left ventricular function. Premature failure of the Hancock pericardial valve resulted in an unacceptable rate of valve-related complications.

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Computerized Potential Distribution Mapping: A New Intraoperative Mapping Technique for Ventricular Tachycardia Surgery

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This study evaluated potential distribution mapping as a method for localizing the site of origin of ventricular tachycardia (VT). In contrast to conventional activation time maps, potential distribution maps require less editing and thus can be more automated and rapidly processed for interpretation of multiple beats of VT. As a series of potential distribution maps during VT is required for detailed analysis, an on-line computerized system was designed to display potential distribution maps sequentially at 1-ms intervals as a color movie. Potential distribution maps and activation time maps were constructed from 182 epicardial and endocardial unipolar electrodes during 12 episodes of reproducible monomorphic VT in 9 dogs four to six days after experimental myocardial infarction (mean cycle length, 162 ± 21 ms). At the onset of each depolarization during VT, a potential minimum abruptly developed on the surviving epicardium and another on the surviving endocardium of the left ventricle, both immediately adjacent to the subendocardial infarct. These two minima on the initial potential distribution maps corresponded to the sites of earliest epicardial and endocardial activation breakthrough recorded on the activation time maps. These two minima subsequently expanded or moved into the adjacent area and coincided with the spread of activation fronts on the epicardial and endocardial surfaces. Thus, the rapid display of sequential, computerized potential distribution maps of multiple beats of VT provides a dynamic means of identifying the site of origin of VT, and therefore should facilitate intraoperative mapping.

(Ann Thorac Surg 1990;49:649–55)

Intraoperative mapping to localize the site of origin of ventricular tachycardia (VT) guides surgeons in determining the appropriate type of operative procedure and the region to which it should be applied. During the past two decades, activation mapping has been the exclusive method used to localize the site of origin of these arrhythmias [1, 2]. However, the construction of activation time maps (ATMs) frequently requires editing to derive activation times from complex or questionable electrograms. This editing is time-consuming and requires considerable experience. In addition, ATMs only demonstrate local activity at the sites of the recording electrodes. Potential distribution mapping is another method to demonstrate the site of origin of VT [3–5]. Studies involving VT simulated by rapid ventricular pacing have demonstrated that the intramural pacing site can be localized from the endocardial and epicardial surfaces with a high degree of accuracy [4, 5]. This method has several advantages over ATMs. (1) Less editing is required to construct the isopotential map because measurement of potential is unambiguous. (2) Once the baseline is determined, no additional analysis time is required. Therefore, multiple beats of VT can be rapidly displayed. (3) Because unipolar potentials reflect distant activity, intramural sites of origin of VT may be detected from epicardial or endocardial recording electrodes. However, potential distribution mapping has not come into wide use because a series of maps is required to demonstrate the activation sequence. To overcome this disadvantage of potential distribution mapping and make it clinically feasible, an on-line computerized system was designed to display potential distribution maps sequentially as a movie. The purpose of this study was to determine if potential distribution mapping could provide a rapid and dynamic means of localizing the site of origin of VT in dogs with experimental myocardial infarction.

Material and Methods

Definitions and Descriptions

1. Potential distribution maps (PDMs): A PDM constructed from unipolar electrograms at a specific instant in the cardiac cycle demonstrates the distribution of the potential fields. Reference baseline for calculation of the potential at each instant was obtained from the average of five consecutive samples (5 ms) in the flat portion of the late diastolic segment during normal sinus rhythm. A series of maps is required to demonstrate progressive changes of the potential distributions.

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2. Potential minimum: A potential minimum is the nadir of the negative potential area at an instant in the cardiac cycle.
3. Primary potential minimum: A primary potential minimum is the earliest potential minimum reaching −3 mV in a series of PDMs [4, 5].
4. Potential maximum: A potential maximum is the zenith of the potential area at an instant in the cardiac cycle.
5. Activation time maps: An ATM is displayed as a sequence of different activation time zones. A computer program is used to determine local activation times from unipolar tracings. The peak negative derivative ($-dV/dt_{max}$) of the major deflection of the unipolar complex is the time of local activation.

Animal Preparation

Seventeen adult mongrel dogs of either sex weighing 18 to 25 kg were anesthetized with intravenous sodium pentobarbital (30 mg/kg), and mechanical ventilation was maintained by delivering 40% oxygen from a volume-cycled respirator (Bennett MA-1) through a cuffed endotracheal tube. After antibiotic prophylaxis (600,000 U of penicillin G administered intramuscularly), the chest was opened through a left thoracotomy in the fourth intercostal space and the heart was suspended in a pericardial cradle. The left anterior descending coronary artery was isolated immediately proximal to the first diagonal branch. A snare was lowered gradually onto a suture surrounding the artery, and partial occlusion was maintained for 20 minutes, followed by complete occlusion. The snare was released after two hours of total occlusion, and the myocardium was reperfused. Lidocaine hydrochloride (2 mg/kg) was administered intravenously immediately before coronary occlusion and just before reperfusion. Then, the chest was closed in layers and the dogs were allowed to recuperate with only postoperative antibiotics given (600,000 U/day of penicillin G administered intramuscularly).

Four to six days after myocardial infarction, the animals were reanesthetized and ventilated in the same fashion as described. A polyethylene catheter for monitoring arterial pressure was placed in the left femoral artery. A right thoracotomy was performed through the fifth intercostal space. Both venae cavae were encircled with tapes for control of these vessels, and the azygos vein was ligated and divided. The heart was exposed and suspended in a pericardial cradle. A sanguinous priming solution and pediatric oxygenator (Model H-400, William Harvey) were used for cardiopulmonary bypass. After heparinization (0.1 mg/kg) and direct cannulation of both venae cavae, normothermic cardiopulmonary bypass was instituted at a flow rate adequate for maintenance of a mean atrial pressure of 60 to 80 mm Hg. The interatrial groove was dissected to expose the septal portion of the left atrium. The heart was arrested with electrically induced ventricular fibrillation, and a standard left atriotomy was performed. A three-dimensional mold containing multiple endocardial electrodes was introduced into the left ventricle through the left atriotomy, thus avoiding the need for a left ventriculotomy. After a right atriotomy, a shaped, multielectrode endocardial mold was introduced into the right ventricular (RV) cavity. Sinus rhythm was restored by direct-current cardioversion. The entire epicardial surface of the heart was covered with a multielectrode epicardial mold. A bipolar electrode was sewn on the right ventricular outflow tract for both introducing ventricular tachycardias and recording reference electrograms. A common reference electrode for all cardiac unipolar electrograms was sewn on the right chest wall at a distance of at least 15 cm from the heart. Placement of the electrode in this position minimized electrical noise. Arterial blood gases and serum hematocrit levels were monitored at 15-minute intervals throughout each study. Sodium bicarbonate was administered as needed to maintain an acid-base equilibrium within normal limits at all times. Serum potassium levels were maintained between 3.7 and 4.5 mEq/L.

Electrophysiological Studies

Under normothermic total cardiopulmonary bypass, programmed electrical stimulation was performed to induce ventricular tachyarrhythmias using a programmable stimulator (model DTV-101, Bloom). Pacing was performed at a pulse width of 2 ms at twice late diastolic threshold current. A train of eight paced beats (S_1s) at a basic cycle length of 300 ms was delivered, after which double premature stimuli (S_2 and S_3) were introduced at varying coupling intervals. The S_2–S_3 interval was lowered by 5-ms decrements until noncapture of S_3 resulted, at which time the S_1–S_2 coupling interval was narrowed by 5-ms decrements. This sequence was repeated until either the heart became refractory to the S_2 stimulus, ventricular fibrillation ensured (followed by direct-current cardioversion), or polymorphic or monomorphic ventricular tachycardia appeared. If the S_2 stimulus consistently failed to capture the ventricle, the pacing protocol was repeated at a basic cycle length of 250 ms. If ventricular tachycardia was not induced at one site, several alternative pacing sites were used.

Epicardial and Endocardial Electrodes and Mapping System

Sixty-one silver bead unipolar electrodes, 2 mm in diameter, were positioned (15 mm apart) in 12 arrays on the inside of an epicardial mold (Fig 1A). Based on the epicardial structure of a normal canine heart, this mold was made of 5-mm thick foam rubber so that it could envelop the entire epicardial surface of the ventricles. The elasticity of the foam rubber maintained optimal contact between the unipolar electrodes and the epicardial surface during both systole and diastole.

The endocardial molds for the left and right ventricles were also made of foam rubber. The RV mold, containing 60 unipolar electrodes, was designed to conform to the irregular internal anatomy of the RV cavity (Fig 1B). The right ventricular electrode was introduced into the RV cavity through the tricuspid valve annulus. The left ventricular mold (Fig 1C) was ellipsoid, 6 cm long and 3 cm across, with 61 unipolar electrodes evenly mounted (10 mm apart) in 12 arrays on its surface. Under normothermic cardiopulmonary bypass during electrically induced ventricular fibrillation, these electrode-containing molds were introduced into the RV and left ventricular (LV) cavities through the tricuspid and mitral valve annuli. The elasticity of these endocardial molds maintained optimal contact between the electrodes and the endocardial surface during both systole and diastole. These epicardial and endocardial molds standardized 182 unipolar electrode positions on the epicardial and endocardial surfaces (Fig 2).

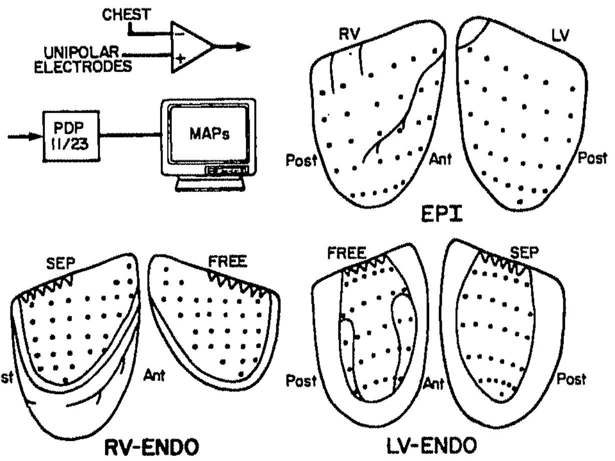

Fig 2. One hundred and eighty-two epicardial and endocardial electrode positions standardized by the epicardial and endocardial molds. (Ant. = *anterior;* ENDO = *endocardium;* EPI = *epicardium;* FREE = *free wall;* LV = *left ventricle;* Post. = *posterior;* RV = *right ventricle;* SEP = *septum.)*

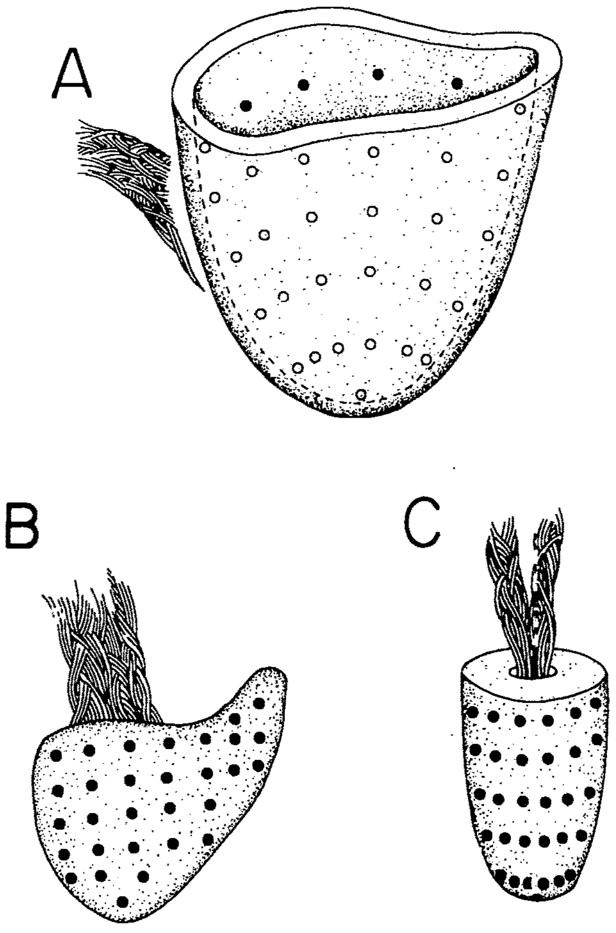

Fig 1. Epicardial and endocardial molds containing 182 2-mm silver bead unipolar electrodes. These molds were made of foam rubber to conform to the epicardial and endocardial structures of the canine heart. (A) Epicardial mold containing 61 electrodes. (B) Right ventricular endocardial mold containing 60 electrodes. (C) Left ventricular endocardial mold containing 61 electrodes.

All signals from each unipolar electrode were connected to the differential amplifiers at an input impedance of 10^{12} ohms and a frequency response of 0.05 to 1,000 Hz. Data were recorded in three sets of 61 electrograms, in addition to the lead II electrocardiogram and bipolar reference electrode electogram. Because the current mapping system was capable of simultaneously recording 64 channels, three time-aligned sets of data using the bipolar reference electrogram and lead II electrocardiogram were required to construct the entire epicardial and endocardial maps from 182 unipolar electrograms. Time-aligned analysis for polymorphic VTs was impossible because of the changing rate and morphology of these arrhythmias. Therefore, the analysis of polymorphic VTs was excluded from this study. However, with a newer 250-channel data acquisition system, polymorphic VT can be analyzed using this technique without any additional processing beyond what is needed to analyze monomorphic tachycardias. A 64-channel analog-to-digital converter digitized the unipolar data at a rate of 1,000 samples/s. A PDP 11/23 PLUS computer stored the data and displayed the waveforms. The input range of the analog-to-digital converter was ± 10 V. An amplifier gain of 500 was used for giving an input signal range of ± 20 mV. Potential distribution maps for a selected window before and during early ventricular depolarization were automatically produced from the computer analysis of all 182 unipolar potentials referenced to the baseline T-P segments of each electrogram. If no baseline could be determined owing to the occurrence of a rapid VT, two other techniques for determining baseline were tested. Baseline was determined from the T-P segment during normal sinus rhythm for each electrogram, and the baseline was also determined for each electrogram by shorting the inputs of each amplifier to ground. Evaluation of PDMs during VT using all three techniques gave equivalent results. In addition, ATMs were similarly produced from the activation times derived from the same unipolar signals.

Both ATMs and PDMs were displayed as smooth, computer-generated isochronous or isopotential maps. As construction of contour lines required considerable time, we used a more rapid "tile map" technique [11] intraoperatively in which each of the electrode points divides the surface of the heart into polygonal regions. Each polygon tile color represented a potential level. More than 1,000 (1,000 ms) PDMs were generated with tile map technique within five minutes after acquisition of the data. Subsequently, a movie of the tile map was displayed by updating the color map for each electrode potential at each millisecond.

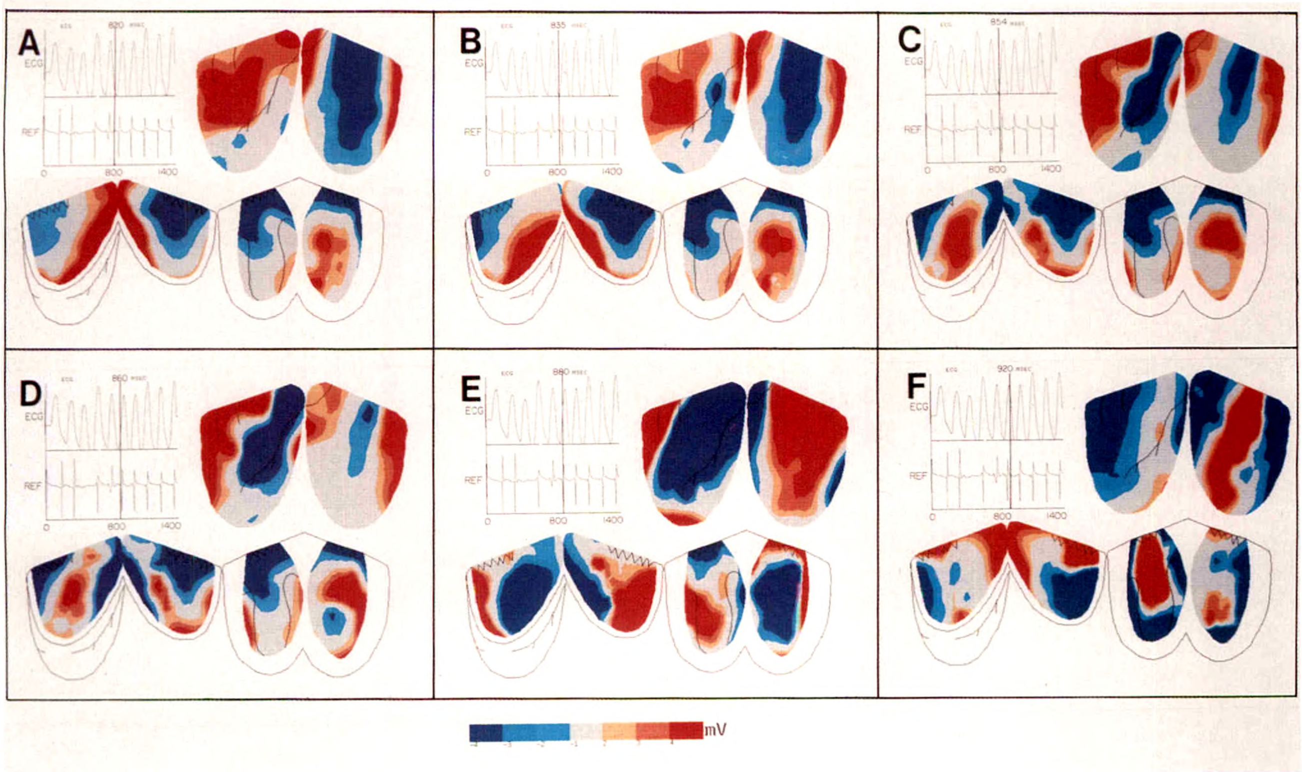

Fig 3. Epicardial and endocardial potential distribution maps during monomorphic ventricular tachycardia. Each panel (A–F) illustrates the computer-generated color isopotential maps of the epicardial and endocardial surfaces. The standard lead II echocardiograph (ECG) *and bipolar reference electrogram* (REF) *are displayed in the left upper corner of each panel. The first, second, and third QRS complexes are* S_1, S_2, *and* S_3 *of a paced train, and the fourth QRS complex is the first beat of the induced monomorphic ventricular tachycardia. The vertical line and number on the ECG tracing indicate the time of each potential distribution map from the beginning of the ECG window. The scale is shown at the bottom of the figure. Negative potentials are idicated by* blue areas, *positive potentials by* red areas, *and near-neutral potentials by* gray areas.

At the end of each experiment, the heart was excised with the endocardial and epicardial electrode molds still in place. The exact locations of the pacing and recording electrodes were verified. The heart was then sliced and stained with triphenyl tetrazolium chloride to define the extent of the infarction and the relationship to the recording electrodes.

All animals received humane care in compliance with the "Principles of Laboratory Animal Care" formulated by the National Society for Medical Research and the "Guide for the Care and Use of Laboratory Animals" (NIH publication No. 85-23, revised 1985). In addition, the experimental protocol was reviewed and approved by the Washington University Committee for the Humane Care of Laboratory Animals.

Results

Characteristics of Induced Ventricular Tachycardia

Seventeen dogs were studied four to six days after myocardial infarction. Monomorphic VT was induced in 9 of 17 dogs. Six of these dogs had one monomorphic VT and 3 dogs had two morphologically distinct VTs. The mean VT cycle length was 162 ± 21 ms (range, 121 to 205 ms). These VTs terminated spontaneously or were terminated by premature ventricular pacing two to five minutes after onset. In 3 of the remaining 8 dogs, polymorphic VT was induced and deteriorated into ventricular fibrillation. No VT was induced in 5 dogs. All 12 episodes of monomorphic tachycardia demonstrated an epicardial reentry pattern in regions overlying the infarct as determined by ATMs and PDMs.

Potential Distribution Maps During Monomorphic Ventricular Tachycardia

Because of the similarity of results, a series of PDMs during monomorphic VT will be presented for a single dog (Fig 3). In the standard lead II electrocardiographic (ECG) tracing, the first, second, and third QRS complexes are S_1, S_2, and S_3 of the paced train; and the fourth QRS complex is the first beat of the induced monomorphic VT. The vertical line and number on the ECG tracing indicate the time of each PDM from the beginning of the ECG window. The potential scale is displayed at the bottom of the figure. Blue regions indicate negative potentials and red regions indicate positive potentials. Near-neutral potentials are indicated by the gray areas. Figure 3A shows the potential distributions at 820 ms after the beginning of the ECG window. At this instant, the majority of the epicardial and endocardial unipolar electrograms were in the repolarization phase (ie, T wave) of the second beat of the induced VT. Therefore, the potential minima are the results of the negative T waves present on the lateral LV epicardium and on the base of the LV and

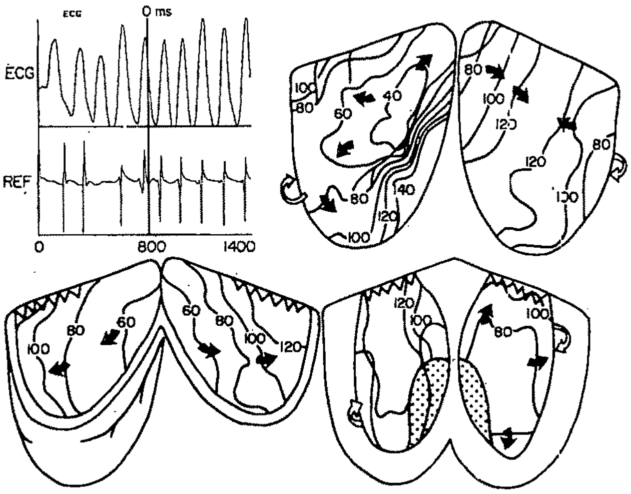

Fig 4. Activation maps recorded from the same animal and during the same beat as in Figure 3. Zero milliseconds for reference time of the activation maps is selected at 800 ms from the beginning of the echocardiographic (ECG) window. The earliest activation site is the paraseptal area and corresponds to the site of the primary potential minimum on the potential distribution maps in Figure 3B. The shaded area *on the left ventricular endocardium represents the infarction zone. (REF = bipolar reference electrogram.)*

RV endocardium. The potential maxima are the results of the positive T waves observed on the epicardium of the RV and on the outflow tract of the RV endocardium. Later on, with the completion of local repolarization, these minima and maxima formed by the T waves slowly diminished the negative and positive areas.

At 835 ms (Fig 3B), the beginning of the third QRS complex of the induced VT, a primary potential minimum abruptly developed in the paraseptal area of the epicardium just adjacent to the subendocardial infarction. At the same instant, there was little change in the epicardial and endocardial potential distributions except for the disappearance of a potential maximum on the RV epicardium. At 854 ms (Fig 3C), the potential minimum on the paraseptal epicardium expanded parallel to the left anterior descending artery. At this instant, the endocardial potential distribution suddenly changed, with a primary potential minimum of the RV endocardium developing anteriorly at the outflow tract. There was little change on the LV endocardium. At 860 ms (Fig 3D), while both epicardial and RV endocardial minima were expanding, another primary potential minimum of the LV endocardium abruptly developed on the LV septum adjacent to the preexisting potential maximum. This site of LV potential minimum was just adjacent to the subendocardial infarction. Subsequently, all epicardial and endocardial potential minima expanded or moved into the adjacent areas, with developing positive areas adjacent to these minima (Figs 3E, 3F). This pattern repeated for successive beats of VT.

Comparison Between Potential Distribution Maps and Activation Time Maps

On the ATMs (Fig 4) recorded from the same animal and during the same beat as that shown in Figure 3, the earliest activation breakthrough was observed on the paraseptal area of the epicardium including the site of epicardial primary potential minimum in the PDMs (Fig 3B). Right ventricular and LV endocardial activation breakthroughs also corresponded to the sites of endocardial primary potential minima following the epicardial primary potential minimum (Fig 4 vs Figs 3C, 3D). A comparison of ATMs and a series of PDMs showed that the potential minima during ventricular depolarization expanded or moved into the adjacent areas, concomitant with the spread of activation fronts at the epicardial and endocardial surfaces (Fig 4 vs Figs 3B–F). For example, a comparison of Figure 3E and Figure 4 shows that the demarcation lines of the negative potential fields in the PDMs at 880 ms are in agreement with the locations of the 80 ms isochrone (880 ms from the beginning of the window) in the ATMs. Although activation breakthroughs in ATMs and primary potential minima in PDMs did not occur simultaneously, the sites of the activation breakthroughs corresponded to those of the primary potential minima. The breakthroughs of ATMs were more widespread than the primary potential minima of PDMs, which occurred epicardially over the subendocardial infarction of endocardially adjacent to the infarction of the left ventricle (Fig 5).

Comment

The advent of direct surgical procedures for the treatment of refractory ischemic VT has led to a substantial improvement in the treatment of patients with these life-threatening arrhythmias [6]. As electrophysiologically guided operation has resulted in a low postoperative inducibility and recurrence of VT, intraoperative electrophysiological mapping is essential for definitive surgical treatment [7, 8]. The primary objective of intraoperative mapping is to localize the site of origin of VT, because ablation of the earliest activation site has been shown clinically to interrupt VT [7, 8]. During the past two decades, epicardial, endocardial, and intramural activation time mapping have been used exclusively for identifying such sites of origin. Hand-held electrodes have been used to record multiple electrograms sequentially to derive the local activation time. However, the ATMs constructed by this technique are less accurate in demonstrating the activation sequence, because these are composite isochrones constructed from local activation times in different beats of VT. More accurate maps can be constructed using multiple simultaneous electrical recording systems. With the use of an increasing number of electrodes and amplifiers, a computer system is required to process the data rapidly [9, 10]. However, the construction of ATMs frequently requires editing to derive activation times from complex or questionable electrograms. This editing is time-consuming and requires considerable experience. Additionally, intuitive judgment is used in interpreting the activation time data, whether they are displayed numerically or as isochrones. As a result of the time required to process the data, usually only one beat of the tachycardia is analyzed, precluding the use of map-

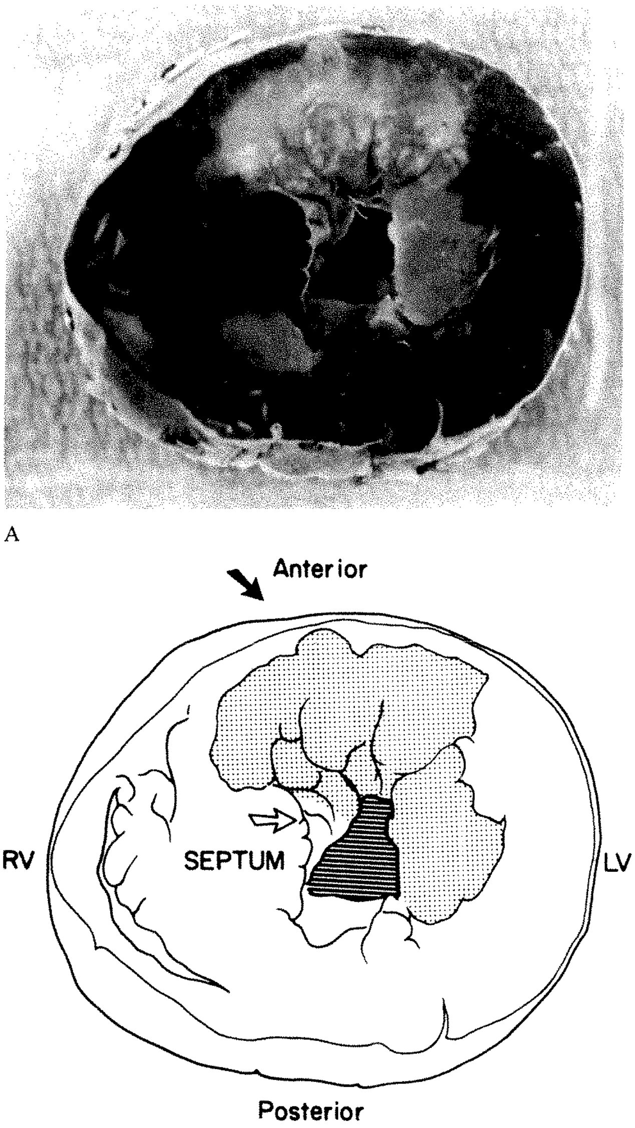

Fig 5. Photograph (A) and diagram (B) of a cross-section of the canine heart from which data are presented in Figures 3 and 4. (A) The section is stained with 2,3,5-triphenyl tetrazolium chloride solution. In (B), the shaded area *represents necrotic tissue from myocardial infarction. The* black arrow *indicates the site of the epicardial primary potential minimum demonstrated in Figure 3B. The* white arrow *indicates the site of the left ventricular primary potential minimum in Figure 3D. (LV = left ventricle; RV = right ventricle.)*

guided operation in polymorphic VT. In contrast to ATMs, PDMs require less editing to construct the isopotential contours, because the measurement of potential distributions is unambiguous, and require no additional analysis time to examine multiple beats of the tachycardia. Therefore, potential distribution mapping can be more automated and rapidly processed for interpretation. Another theoretical advantage is that unipolar potential maps can reflect distant activation events. Thus, epicardial and endocardial maps could theoretically detect early intramural wavefronts, whether in the ventricular free walls or septum, before the actual emergence of activation at these surfaces. Previous data on paced intramural ventricular beats indicate that not only the site but also the intramural depth could be approximated from analysis of the PDMs from the ventricular surfaces bordering the site of stimulation [5]. With uniform conduction, the emergence of the wavefront would be expected to be at the site on the endocardial or epicardial surface closest to the point of origin. The presence of nonuniform activation due to the infarction or unidirectional block during reentrant ventricular rhythms could spatially dissociate the site of intramural depolarization and endocardial or epicardial breakthrough sites. Because PDMs reflect distant and local activity, electrode spacing is not as critical as it is for ATMs, which only represent local events. In addition, potential distributions recorded in areas of infarcted tissue (or areas with overlying thick fat pads) provide information to localize the origin of the VT. When constructing ATMs, these areas provide little information as no activation time can be assigned. In this situation, PDMs could prove to localize the intramural origin more accurately than endocardial or epicardial activation maps as the surface potentials would reflect intramural and distant activation.

Epicardial, endocardial, and intramural potential distribution during ventricular pacing have been studied previously by several investigators [3–5]. It has been well documented that the earliest potential minimum develops in an area close to the pacing site (earliest activation site) after the stimulation and that this minimum subsequently expands and moves to a distant area as it increases in negativity. Thus, the earliest potential minimum with low amplitude should represent the area closest to the earliest activation site [5]. We previously demonstrated that the earliest potential minimum reaching −3 mV is defined as a primary potential minimum that corresponds to the earliest activation site. Using the same definition for a primary potential minimum in this study, potential distribution mapping was evaluated as a method to localize the site of origin of VT. As described in the Results section, the epicardial primary potential minimum occurred above the subendocardial infarction and the LV-endocardial primary potential minimum occurred adjacent to that infarction. These minima corresponded to the sites of activation breakthrough on the ATMs. Because an instant of $-dV/dt_{max}$ in the unipolar electrogram (activation time) usually did not coincide with the instant reaching −3 mV (primary potential minimum), activation breakthrough and primary potential minimum did not appear simultaneously. However, regardless of whether primary potential minimum precedes or follows activation breakthrough, it localizes the earliest activation site. Therefore, potential distribution mapping can serve the primary purpose of intraoperative mapping to localize the site of origin of VT. Furthermore, breakthrough on the ATMs appeared as a more widespread area than the primary potential minimum.

One problem with viewing the PDMs statically is that there are often large areas of negativity. These areas are associated with the T waves, and when the maps are viewed dynamically in rapid succession, they are easily differentiated from the negativity associated with activation because they change much more slowly. However, the potential distribution mapping technique could be improved if the T waves were filtered out of the signal.

Spach and Barr [3] demonstrated that the demarcation line separating positive and negative potential (0 mV) shows good agreement with the location of the activation excitation wave. Our study demonstrated that the boundary of the negative potential field less than −3 mV at each instant coincides with the location of the activation wave. Rapid display of a series of PDMs could visually demonstrate the dynamic change of the activation fronts during VT. Therefore, we designed an on-line computer system to display PDMs sequentially as a movie.

In conclusion, computerized potential distribution mapping provides a rapid means for the mapping of multiple beats of VT for the purpose of identifying the sites of origin of VT and also a dynamic means of demonstrating the activation sequence of these arrhythmias. Therefore, this intraoperative mapping technique should facilitate intraoperative mapping in patients undergoing operations for ventricular tachycardia.

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Free Arterial Graft as Internal Mammary Artery–Y Complex

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Because of excellent surgical results obtained with in situ internal mammary artery grafts, recent efforts have been made to extend the use of arterial conduits. In this study 2 cases of coronary revascularization using free internal mammary artery and gastroepiploic artery grafts as an internal mammary artery–Y complex are described. We believe that complete revascularization with only arterial conduit can be achieved more widely by using this technique.

(Ann Thorac Surg 1990;49:656–8)

This report describes the use of internal mammary artery and gastroepiploic artery grafts as an internal mammary artery (IMA)–Y complex for coronary artery bypass grafting. This procedure was used in 2 patients in whom standard grafting techniques were not feasible.

Patient 1

A 62-year-old man with a history of pleuritis was admitted with the diagnosis of two-vessel disease. The left anterior descending coronary artery was totally obstructed at the proximal portion and the collateral flow was from the right coronary system. There was also 90% stenosis at the posterolateral branch. Bypass grafting using bilateral IMAs was planned, but the left IMA adhered to the chest wall firmly at the sternal notch and was difficult to separate. The left IMA had no length to reach the circumflex system if used in situ; therefore, we decided to use it as a free graft. Before cardiopulmonary bypass was begun, the proximal anastomosis was made opposite to the IMA as a Y anastomosis using 8-0 polypropylene continuous suture technique. The distal anastomoses were made under cardioplegic standstill as usual; in situ right IMA bypassed the left anterior descending coronary artery and a free graft of the left IMA bypassed the posterolateral branch. The postoperative course was not eventful. Coronary angiography performed 33 days after operation revealed good patency of both grafts, and the patient was discharged with no sign of ischemia.

Patient 2

A 74-year-old man with diabetes mellitus and coronary disease was admitted. There was 99% organic stenosis at the most proximal portion of the left anterior descending coronary artery and 75% stenosis in the large diagonal branch. Considering complication of diabetes mellitus, the use of an arterial graft was planned and accomplished; the left IMA bypassed the left anterior descending coronary artery and a free graft of the right gastroepiploic artery bypassed the diagonal branch (Fig 1). The left IMA flow was 100 mL/min and its diameter was 2 mm. There was severe sclerotic change at the root of the ascending aorta by palpation, and aortic anastomosis seemed to be hazardous. Before cardiopulmonary bypass was begun, the free gastroepiploic artery was anastomosed to the left IMA in situ according to the same method as used in patient 1, and then the distal anastomoses were constructed under cardioplegic standstill. The patient's postoperative course was not eventful. Coronary angiography performed 30 days after operation revealed good patency of the both grafts (Fig 2). Left ventricular ejection fraction improved from 0.50 preoperatively to 0.77 postoperatively.

Comment

Because of the excellent late patency obtained from in situ IMA grafts, efforts have been made to extend the use of the IMA conduit [1, 2]. Use of bilateral IMAs and sequential technique are becoming popular [3], and there have been some reports of IMA use as a free graft [4–7].

Recently gastroepiploic artery grafting has become popular [8–10], and the use of the gastroepiploic artery as a free graft has been reported by Lytle and associates [9] and Mills and co-workers [10]. However, most proximal anastomoses are made in the aortocoronary position to the ascending aorta. It may be hazardous to anastomose thin arterial grafts to the thick aorta, and technical problems with the proximal aortic anastomosis have been implicated as a cause of early graft failure [11, 12]. Therefore, we believed that proximal anastomosis of free arterial graft should be made on in situ IMA and IMA–Y complex.

IMA grafting is necessary in patients with diseased or absent long and short saphenous vein: however, it is frequently not possible anatomically to reach arteries on the posterior surface of the heart with either IMA, whether as a pedicle or as a free graft of aortocoronary position. In addition, we occasionally encounter a firmly

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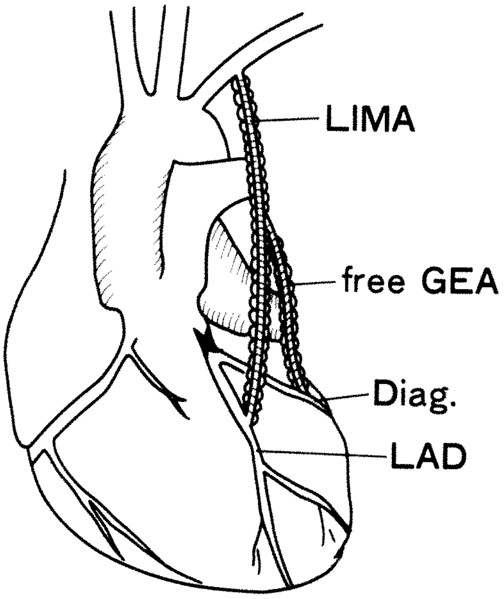

Fig 1. Coronary revascularization using free gastroepiploic artery as internal mammary artery–Y complex. (Diag. = *diagonal branch;* GEA = *gastroepiploic artery;* LAD = *left anterior descending coronary artery;* LIMA = *left internal mammary artery.)*

adhered IMA. In such patients, use of the free arterial graft as IMA–Y complex should be implemented. Sauvage and associates [3] proposed IMA–Y technique for reaching posterior marginal circumflex branches and the right coronary artery. This technique is also beneficial when performing coronary artery bypass on patients with diffuse atherosclerotic ascending aorta to prevent cross-clamping of the aorta [10].

A major concern about this procedure is whether attached IMA is capable of supplying enough blood to the larger areas of the ischemic myocardium as Tector and colleagues [1] mentioned. In the patients reported here, free flow of attached IMA was over 100 mL/min and the diameter of IMA was 2.0 mm. Postoperative stress tests showed no sign of ischemia in either patient. The left ventricular contractions demonstrated on the postoperative ventriculograms vastly improved in the anterior, lateral, and posterior walls. From our experience, IMA–Y technique should be indicated in patients whose IMAs are large and whose IMA free flow is more than 100 mL/min.

We believe that complete revascularization with only arterial graft (IMA, gastroepiploic artery) can be achieved more widely by using this technique of free arterial graft as IMA–Y complex.

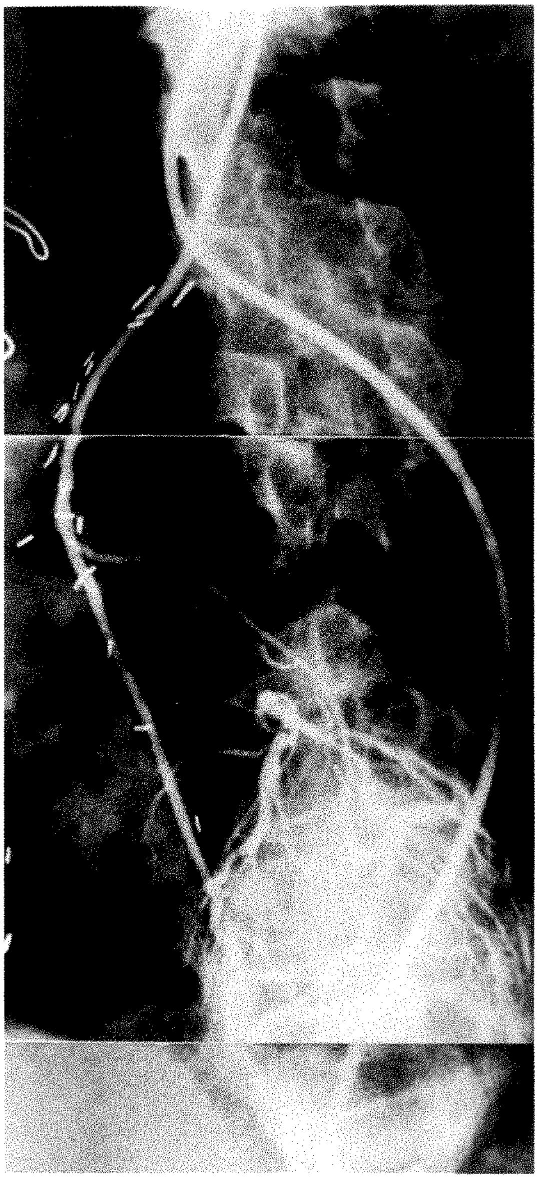

Fig 2. Postoperative coronary artery angiography shows good patency of both in situ internal mammary and free gastroepiploic artery grafts.

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REVIEW OF RECENT BOOKS

New Trends in Heart Transplantation (Bibliotheca Cardiologica, No. 43. Series Editors, J. J. Kellermann and Tel Hashomer)

Edited by E. Wolner, A. Laczkovics, and M. Havel
Basel, Karger, 1988
167 pp, illustrated, $97.50

Reviewed by Charles J. Mullany, MB, MS

This volume consists of 26 papers presented to the First Vienna Symposium on New Trends in Heart Transplantation, September 1987. The majority of these papers (21) are short reports of the clinical immunological experiences of the major European transplant centers.

The volume is divided into three sections. The first section deals with various methods of immunosuppression, commencing with a short review of current immunosuppressive techniques. The other chapters deal with clinical reports of variations in methods of immunosuppression, including experiences with triple and quadruple therapy, the use of monoclonal OKT3, and the management of acute rejection using oral steroids.

The second section deals with various aspects of cyclosporine pharmacology including controversies in cyclosporine measurements, the determination of its metabolites, and a discussion of their possible role in immunosuppression. Included in this section is a report of the use of the serine proteinase inhibitor aprotinin (Trasylol) to prevent postoperative hemorrhage after cardiac operations. Inclusion of this article in the book seems inappropriate.

The final section deals with cardiac rejection. There is an excellent review chapter by Billingham on the interpretation and pitfalls of endomyocardial biopsies in the diagnosis of cardiac rejection. Other chapters in this section discuss the role of noninvasive techniques in diagnosing acute rejection. These include the role of antigen-unspecific hormonelike factors (lymphokines) in the regulation of allograft rejection, and the identification of electrophysiological, cytoimmunological, and hemodynamic variables in cardiac rejection. However, it is clear that endomyocardial biopsy remains the most reliable tool for the diagnosis of rejection. This section also consists of a short review article on the role of monoclonal antibodies for the diagnosis and treatment of infection.

This volume, apart from the three useful review articles on immunosuppressive techniques, endomyocardial biopsy, and monoclonal antibodies, covers in a very superficial way the very specialized areas of immunology and cyclosporine pharmacology related to cardiac transplantation. To those surgeons seeking a detailed overview of current practice and controversies in heart transplantation, this book would appear to be totally inadequate. To those well versed in detailed aspects of immunology and cyclosporine chemistry, these various chapters, or in some instances case reports, may prove valuable as a reference for current work. Although this book would be a useful library source, I would not recommend it as an essential text for the cardiothoracic surgeon's reference library.

Rochester, Minnesota

Tricuspid Regurgitation With Postinfarction Ventricular Septal Defect

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Severe tricuspid regurgitation became apparent in 2 patients who had acute inferior myocardial infarction after repair of a ventricular septal defect. Subsequent tricuspid valve insertion was performed successfully in 1 patient. The pertinent literature is reviewed.

(Ann Thorac Surg 1990;49:659–60)

Ventricular septal defect after acute myocardial infarction occurs in about 1% to 2% of patients [1]. Acute inferior wall infarction complicated by ventricular septal rupture or tricuspid regurgitation is extremely rare [1, 2]. This report describes our experience with 2 patients who were seen with tricuspid regurgitation after surgical repair of ventricular septal defect resulting from acute inferior wall myocardial infarction.

Patient 1

A 65-year-old man was admitted with an evolving acute inferior wall myocardial infarction. The next day he became hypotensive and a new loud systolic murmur developed. Insertion of a Swan-Ganz catheter revealed high central venous pressure with prominent V wave (25 mm Hg), pulmonary artery pressure of 35/20 mm Hg, and 35% step-up in oxygen saturation from the right atrium to the main pulmonary artery. Cardiac catheterization showed a posterior ventricular septal defect, inferior wall akinesis, total occlusion of the right coronary artery, and a normal left coronary artery. The patient remained hemodynamically unstable; an intraaortic balloon pump was inserted and an emergency operation was performed.

At operation the infarction involved most of the diaphragmatic surface of both right and left ventricles. High left ventriculotomy through the infarction revealed extensive necrosis of the septum and a septal defect near the base of the septum measuring approximately 2 × 4 cm. Much of the posterior papillary muscle was infarcted, but otherwise the tricuspid valve and its supporting mechanism appeared to be normal. Replacing the tricuspid valve was not considered because it was believed that a passive tricuspid competence would result and be efficient. The septum and the right and left ventricular portions of the infarct were generously debrided with care taken to avoid the coronary sinus. The resulting septal defect was closed with a double or "sandwich" patch method using Teflon felt brought out on the right ventricular side of the infarctectomy. The resulting high inferior wall defect was closed by the method of Daggett and co-workers [3] using a composite patch of Teflon felt and pericardium. On the fourth postoperative day, after the removal of the intraaortic balloon pump, a grade 2/4 systolic murmur was heard along the right sternal border. However, the patient remained hemodynamically stable, although he did require prolonged respiratory assistance for a period of 14 days. He was discharged in satisfactory condition on the 26th postoperative day.

After discharge, the patient did well for approximately 3 months, but progressive dyspnea, cough, anorexia, increasing abdominal girth, and a pulsatile liver began to develop. Repeat cardiac catheterization demonstrated 4+ tricuspid regurgitation with a trivial residual ventricular septal defect. At reoperation the tricuspid annulus was grossly dilated, admitting five fingers, and the posterior papillary muscle was scarred and attenuated. There was a 1-cm hole in the anterior leaflet, the posterior leaflet was adherent to the septal and inferior wall patches, and several chordae of the anterior leaflet were torn. The entire tricuspid mechanism was left intact and a No. 31 porcine bioprosthesis was inserted. The residual 1 × 1-cm septal defect near the apex was repaired incorporating the previously placed ventricular wall patch. Postoperatively, the patient did well, was extubated 24 hours later and was discharged. At 34 months after operation, the patient remains in New York Heart Association class II.

Patient 2

A 70-year-old man was seen with an acute inferior wall infarction and received immediate thrombolytic therapy. Cardiac catheterization revealed a totally occluded right coronary artery, a severely stenotic left anterior descending coronary artery, and left ventricular inferobasilar hypokinesis. He was treated medically, did well, and was discharged. Three weeks after infarction he was seen again with severe chest pain associated with dyspnea and hypotension. A new systolic murmur was heard. Right heart catheterization demonstrated a 10% step-up in oxygen saturation from the right atrium to the right ventricle. A diagnosis of ventricular septal defect was made, an intraaortic balloon was inserted, and the patient was taken to the operating room.

Repair of a posterior ventricular septal defect was carried out with a Teflon felt patch, the biventricular wall defect was reconstructed with a Dacron patch, and the left anterior descending coronary artery was bypassed using a vein graft. After discontinuation of the intraaortic balloon

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pump on the third postoperative day, a grade 3/6 systolic murmur became audible along the right sternal border. Over the ensuing weeks, clinical evidence of progressive tricuspid regurgitation with right ventricular failure developed. A radionuclide scan showed tricuspid regurgitation with massive enlargement of the right atrium and ventricle in addition to a small residual septal defect and a right ventricular ejection fraction of 21%. Postoperatively, poor mentation, hemiparesis, and pneumonia developed. The family refused further operation and the patient died one month after repair. Permission for autopsy was denied.

Comment

Ventricular septal defect after acute right coronary occlusion is uncommon [4]. Similarly, tricuspid regurgitation after infarction involving the right ventricle has been infrequently reported. A review of its incidence by Lader and associates [2] in 1983 disclosed only 7 such patients with one documented rupture of a tricuspid papillary muscle. Right ventricular damage due to infarction has been increasingly recognized as an important clinical entity with potentially grave consequences [5]. In fact, patients with postinfarction ventricular septal rupture who have right ventricular dysfunction tend to experience cardiogenic shock and have a much worse prognosis for survival than those who do not [6, 7].

The occurrence of ventricular septal defect and tricuspid regurgitation after inferior wall myocardial infarction, to our knowledge, has previously been described only twice in the past [2, 8]. Of interest, the first successful repair of postinfarction ventricular septal defect by Cooley and colleagues [8] in 1957 was done in the setting of an inferior wall infarction. At operation, a regurgitant jet suggestive of tricuspid regurgitation was palpable in the right atrium. Another patient reported by Lader and co-workers [2] had a similar inferior wall infarction and progressive right ventricular failure. Cardiac catheterization revealed total occlusion of the mid-right coronary artery without substantial stenosis in the left coronary artery, a ventricular septal defect, and an elevated right atrial pressure with a V wave of 26 mm Hg. At operation, the posterior papillary muscle of the tricuspid valve was found ruptured and was replaced with a porcine bioprosthesis; the septal defect was closed with a Dacron patch. The postoperative course was uneventful.

Our patients also had total occlusion of the right coronary artery, and inferior wall ventricular septal defects developed after infarction. Tricuspid regurgitation very likely existed in patient 1 before the first operation, as a clinically significant right atrial V wave was noted during the Swan-Ganz catheter insertion. The significance of this finding was not appreciated because there were no clinical signs of tricuspid regurgitation due to low cardiac output and shock.

The prognosis after predominant right ventricular infarction is generally good [9], and isolated mild-to-moderate tricuspid regurgitation is usually well tolerated [10]. The progression of tricuspid insufficiency and clinical deterioration in our patients was probably enhanced by the recurrent septal defect and the resulting shunt, which caused increased right ventricular pressure and volume loads and right ventricular dilatation.

In summary, inferior myocardial infarction may cause the combination of ventricular septal defect and tricuspid papillary muscle infarction with or without rupture. The surgeon should be aware of this association and of its severely damaging effects on right ventricular function, and should have a low threshold for replacing the tricuspid valve in the setting of papillary muscle infarction, whether or not there is actual rupture at the time of ventricular septal defect repair. Valve insertion rather than resection and replacement is notably advantageous in preserving right ventricular function.

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Management of Major Tracheal Hemorrhage After Repair of Complex Congenital Heart Defects

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Two patients with complex congenital heart defects (a 4-year-old with transposition of the great arteries, ventricular septal defect, and left ventricular outflow tract obstruction and a 3½-year-old with double-outlet right ventricle, subpulmonary stenosis, and complete atrioventricular septal defect) suffered multiple major hemorrhages from the tracheobronchial tree (28 and 7 bleeding events, respectively). Successful management included tracheostomy, sedation and paralysis, systemic hypotension, and systemic hypothermia for a period of seven days. Both patients survived.

(Ann Thorac Surg 1990;49:661–3)

Major tracheobronchial bleeding in children may be life threatening. It is a rare but usually catastrophic complication after repair of complex congenital heart defects. Although several authors [1–4] describe bleeding from an eroded major vessel as a complication of tracheostomy or prolonged intubation, we were unable to find published reports about bleeding after correction of congenital heart disease.

We have encountered major tracheobronchial bleeding in 2 children. One was a 3-year-old boy who underwent a Rastelli-type repair for transposition of the great arteries, ventricular septal defect, left ventricular outflow tract obstruction with previous Blalock-Hanlon septectomy, and Blalock-Taussig shunt. The second patient had a repair of complete atrioventricular septal defect associated with double-outlet right ventricle and subvalvar pulmonary stenosis. The clinical course of multiple major hemorrhages and their successful treatment form the basis of this report.

Patient 1

This infant underwent a Blalock-Hanlon septectomy and modified Blalock-Taussig shunt at the age of 1 year. At the age of 3 years (July 8, 1988), Rastelli-type repair using a 17-mm aortic homograft was performed. After operation the patient was hemodynamically stable, but because of left phrenic nerve paralysis and chest infection (*Hemophilus influenzae*), weaning from the ventilator was delayed. Three days after extubation, during an episode of coughing, he suffered massive tracheobronchial hemorrhage (approximately 500 mL) with cardiorespiratory arrest. He was reintubated and successfully resuscitated. The same day fever developed, and blood culture grew *Staphylococcus aureus*, for which administration of appropriate antibiotics was started. Several bleeding episodes ranging from small to massive hemorrhage occurred during the next few days.

In an attempt to identify the source of bleeding, a bronchoscopy was performed. Both main bronchi, bifurcation, and lower trachea were clear. No ulcerations were seen. During the procedure the patient had another hemorrhage from the upper trachea. Because of the massive nature of this bleeding the source could not be identified. As the bleeding seemed to be from the upper trachea, we decided to perform a tracheostomy.

After tracheostomy the patient was weaned rapidly to constant positive airway pressure but suffered another major hemorrhage, and full ventilation had to be restarted. Blood culture at this time grew *Pseudomonas aeruginosa*, which was also identified from the sputum. Two-dimensional echocardiography showed possible vegetations on the right ventricular side of the ventricular septal defect patch. It was therefore decided to treat the patient for subacute bacterial endocarditis for 6 weeks with antibiotics.

Another bronchoscopy was performed and an attempt was made to diathermise three ulcerations that were seen near the bifurcation. We were unable to identify any of the ulcerations as the definitive source of bleeding. Subsequently, the patient suffered several major bleeding episodes and two more cardiac arrests from which he was successfully resuscitated. As a last resort we decided on the following therapeutic regimen: tracheostomy, prolonged sedation with morphine drip and paralysis with vercuronium drip, gentle physiotherapy, and aspiration with soft rubber catheters that were not advanced beyond the tip of the tracheostomy tube. Systemic hypotension with sodium nitroprusside (later captopril) and systemic cooling to a central temperature of 34°C were used. Total parenteral nutrition was started.

This treatment was given for eight days. Only one small bleeding episode occurred on day 2, and none occurred during the treatment or after its discontinuation. Although the subacute bacterial endocarditis was kept under control as confirmed by three negative blood cultures and low level of C-reactive protein, weaning from the ventilator was not successful. As he was now clinically in congestive heart failure, he was recatheterized and recurrent ventricular septal defect and aneurysm of the right

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ventricular outflow tract were found. These were repaired and a left paralyzed diaphragm was plicated. No vegetations were seen on the ventricular septal defect patch. His subsequent progress was uneventful. He was weaned from the ventilator, the tracheostomy tube was gradually reduced in size, and he was extubated on October 15, 1988.

During his entire hospital stay, the patient suffered 28 bleeding episodes, 13 of which were major (over 500 mL). He was successfully resuscitated from three cardiac arrests. With the introduction of our therapeutic regimen, there was no more bleeding.

Patient 2

A 3½-year-old girl underwent complete repair of double-outlet right ventricle with infundibular pulmonary stenosis and complete atrioventricular septal defect on December 28, 1988. Her postoperative course was complicated by severe junctional tachycardia that was refractory to treatment. The arrhythmia caused low cardiac output syndrome with oliguric renal failure, which was managed by high doses of furosemide. On the second postoperative day, a high temperature developed and a blood culture was positive. Administration of antibiotics was started. As she was not fully absorbing what was fed to her nasogastrically, additional parenteral nutrition was also given.

Because of her slow progress, cardiac catheterization was performed on February 5, 1989. This confirmed a good surgical result with no hemodynamic defects. The left ventricular function, particularly the diastolic function, was, however, impaired. The patient was treated with digoxin, diuretic agents, dopamine, and afterload reduction. On this regimen she started to improve slowly. She only had a few short episodes of junctional tachycardia but otherwise stayed in regular sinus rhythm.

On the 16th postoperative day she suffered massive tracheal bleeding. This was controlled with difficulty. When she bled again a few hours later, we decided to perform an emergency tracheostomy and to institute the same treatment regimen as described in our previous patient. Shortly after completing the tracheostomy she had another massive bleeding episode, the inflation pressure rose to 60 cm H_2O, and carbon dioxide tension increased to 90 mm Hg despite hand ventilation. She suffered a cardiac arrest and resuscitation lasting 30 minutes was eventually successful.

She was started on the regimen described (paralysis, sedation, cooling, hypotension, parenteral nutrition). She had several small to moderate bleeding episodes within 24 hours of the initiation of treatment, but none subsequently. She was kept on this treatment for seven days. When the paralysis and cooling were stopped, she was quickly weaned from the ventilator and extubated without difficulty. The patient was eventually discharged from the hospital 5 weeks after the operation. She had no neurological sequelae to her low cardiac output and prolonged cardiorespiratory arrest. She altogether suffered seven major bleeding episodes. Now, 8 months later, she is asymptomatic and being given no medications.

Comment

Bleeding from the lung or tracheobronchial tree is a known complication of cyanotic congenital heart defects. In our previous experience, it was a rare but invariably fatal complication occuring mainly in older, severely cyanotic children who had no operation or who underwent only some form of palliative operation. In review of the English-language literature we found reports of bleeding from eroded major vessels as a complication of tracheostomy [1–4] but failed to find a description of massive bleeding after repair of complex congenital heart lesions. We believe that this lack of reports may be due to the fact that such complications were probably fatal in the past.

The cause of bleeding may be difficult to ascertain. We believe that the most likely cause is bleeding from mucosal vessels in the trachea or main bronchi, which are eroded by ulcers caused by the suction catheters or tip of the endotracheal tube after prolonged intubation or ventilation. Arteriovenous malformation in the lung, large bronchial collaterals in cyanotic patients, or mycotic aneurysms in infected patients may be other causes.

The treatment is difficult. If small bleeding occurs, ice-cold saline solution is usually instilled through the endotracheal tube sometimes together with diluted adrenaline. In more massive bleeding, sedation and paralysis of the patient, aspiration of the blood clots, topical cooling, blood replacement, and possibly transfusion of platelets or fresh frozen plasma can be used. If one can establish that the bleeding comes from a particular segment of one lung, lobectomy may be considered. However, we believe that to establish such a source of bleeding with certainty is very difficult. In addition, major lung resection may not be tolerated in a patient after a complex intracardiac operation. Diagnostic bronchoscopy in children who bleed from the tracheobronchial tree is also difficult. In the absence of bleeding, it is generally impossible to ascertain whether one of the ulcerations in the trachea or main bronchi is the one that was responsible for the bleeding. When bleeding occurs, visibility is rapidly lost and aspiration of blood and ventilation through the bronchscope becomes difficult so that the procedure has to be abandoned.

In the 2 patients presented, bleeding did not stop with the conventional treatment. The rationale for our therapeutic regimen was as follows: tracheostomy shortens the length of the tube, diminishes the dead space, and facilitates aspiration of secretions and blood clots. The sedation and paralysis abolishes the coughing reflex that may be the provoking mechanism of the bleeding. The patient is completely still and does not fight the ventilator. Systemic hypotension with sodium nitroprusside and later captopril was used to avoid higher blood pressure, which in cases of arterial bleeding could contribute to the severity of bleeding. We used systemic hypothermia to reduce oxygen requirements of the brain and other vital organs in the event of another hypoxic episode or cardiac arrest and also to provide a safety margin during the controlled prolonged hypotension. The temperature of the humidified air/oxygen in the ventilator was also reduced. Vercuronium infusion was used for paralysis in preference to pancuronium because in some patients the gut function is not impaired and at least partial nasogastric feeding may be possible. This was possible in our second patient, but the first one required total parenteral nutrition during this period of treatment. In addition to these measures only gentle physiotherapy was given and secretions were aspirated with soft rubber catheters. An attempt was made not to pass the catheter beyond the tip of the tracheostomy tube. The length of treatment, eight and seven days, respectively, was selected arbitrarily. The patients bled for the first 24 hours after the institution of this therapy but not afterward. There was no bleeding after discontinuation of this treatment.

In conclusion, we recommend the treatment outlined in this report for patients with major tracheobronchial bleeding after operation for complex congenital heart defects.

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REVIEW OF RECENT BOOKS

Manual of Perioperative Care in Cardiac and Thoracic Surgery
By Robert M. Bojar
Cambridge: Blackwell Scientific Publications, 1989
380 pp, illustrated, $25.00

Reviewed by Jolene Kriett, MD

This book provides an easily readable, concise, and straightforward approach to preoperative and postoperative management of cardiac and thoracic surgical patients based on protocols used at the New England Medical Center. The manual is divided into three main sections. In the first section preoperative and postoperative care of the adult cardiac surgical patient are discussed. The emphasis is placed on practical information regarding the diagnosis and treatment of common postoperative cardiac problems. Topics include patient management in the early postoperative period concentrating on cardiac and pulmonary support, arrhythmia recognition and treatment, and evaluation and management of common complications. The selected topics are approached in a well-organized and concise manner including the rationale for therapeutic decisions based on pathophysiology. In similar straightforward fashion, the second section covers practical information on the management of the pediatric cardiac surgical patient. An introductory synopsis of the anatomy and repair of common congenital heart defects is included with excellent illustrations. The final section of this manual addresses perioperative care of the thoracic surgical patient including preoperative assessment, postoperative care, and management of both postoperative complications and other common thoracic surgical problems. An extensive reference list completes each section for readers interested in more detailed information on the various topics.

Although a great deal of information is covered on a superficial level, the author has succeeded in his stated goal of providing a practical manual for physicians involved in the care of adult and pediatric cardiac and thoracic surgical patients. Such a concise and well-designed book should be extremely useful to medical students and surgical residents rotating on a cardiothoracic surgery service, as well as cardiothoracic surgery fellows.

San Diego, California

One-Stage Repair of Absence of the Aortopulmonary Septum and Interrupted Aortic Arch

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Absence of the aortopulmonary septum, interrupted aortic arch, aortic origin of the right pulmonary artery, intact ventricular septum, and patent ductus arteriosus is a rarely reported association. A 3-year-old boy underwent successful one-stage repair of this constellation of anomalies. A Dacron baffle was used both to close the huge aortopulmonary window and to direct blood to the right pulmonary artery. Type A interrupted aortic arch was repaired by direct anastomosis. Postoperatively, pulmonary artery pressure was less than half systemic pressure.

(Ann Thorac Surg 1990;49:664–6)

Aortopulmonary window is an extremely rare anomaly that may occasionally be associated with interrupted aortic arch. An aortopulmonary window can vary in location and size and has been classified by Richardson and co-workers [1] into types I, II, and III. Type I is usually a relatively small defect between the ascending aorta and main pulmonary artery, immediately above the sinuses of Valsalva. Type II is located on the posterior wall of the ascending aorta at the origin of the right pulmonary artery. In type III the right pulmonary artery arises from the right side of the ascending aorta and there is complete absence of the aortopulmonary septum. Only the fact that there are two separate semilunar valve annuli separated by a thin rim of tissue distinguishes type III from truncus arteriosus.

In 1982, Berry and colleagues [2] described the association of absence of the aortopulmonary septum, aortic origin of the right pulmonary artery, intact ventricular septum, patent ductus arteriosus, and hypoplasia of the aortic isthmus. Of the 8 patients described in their report, including 5 of their own patients and 3 cases collected from the literature, 6 had interrupted aortic arch and 2 had coarctation. Two patients survived surgical repair though 1 died later. In 1 neonate the right pulmonary artery was detached from the aorta and implanted in the main pulmonary artery. A 16-year old child had a tube graft placed from the aortic annulus to the internal orifice of the aortic arch within the original aorta, allowing the right pulmonary artery to be supplied by blood flow around the tube. The interrupted aortic arch was repaired using the left subclavian artery in 1 patient and a Dacron tube in the other. We report successful surgical management of this anomaly using an alternative technique.

A 35-month-old boy weighing 10 kg was referred to Xin Hua Hospital with a history of frequent respiratory infections. He had been noted to have a murmur in early infancy and was taking digoxin.

Physical examination revealed a blood pressure of 100/45 mm Hg in both arms and legs with no evidence of differential cyanosis or clubbing. There was a 2/6 systolic murmur at the left sternal border and a 2/6 diastolic murmur at the apex. Chest roentgenogram revealed increased pulmonary vascular markings and moderate cardiomegaly. Electrocardiography showed sinus rhythm, axis at 90 degrees, and biventricular hypertrophy. Two-dimensional echocardiography revealed type A interrupted aortic arch, patent ductus arteriosus, intact ventricular septum, and a probable aortopulmonary window (Fig 1A). Cardiac catheterization confirmed the echocardiographic findings. Pulmonary artery pressure was systemic.

Surgical Repair

The child was positioned 45 degrees left side up and was prepared and draped to allow both a left thoracotomy and median sternotomy. Through the left thoracotomy the interruption of the aorta was confirmed. Approximately 1.5 cm separated the left subclavian artery from the proximal descending aorta. The ductus was divided and oversewn on its pulmonary artery end. A direct anastomosis was fashioned between the undersurface of the aortic arch and the proximal descending aorta.

A median sternotomy incision was made. The right pulmonary artery was noted to be arising from the right lateral surface of the ascending aorta, which was dilated with a diameter of 25 mm (Fig 2). Cardiopulmonary bypass was established after heparinization. The arterial cannula was positioned in the mid-descending thoracic aorta through the thoracotomy incision and a single venous cannula was placed in the right atrium through the sternotomy incision. The right pulmonary artery was

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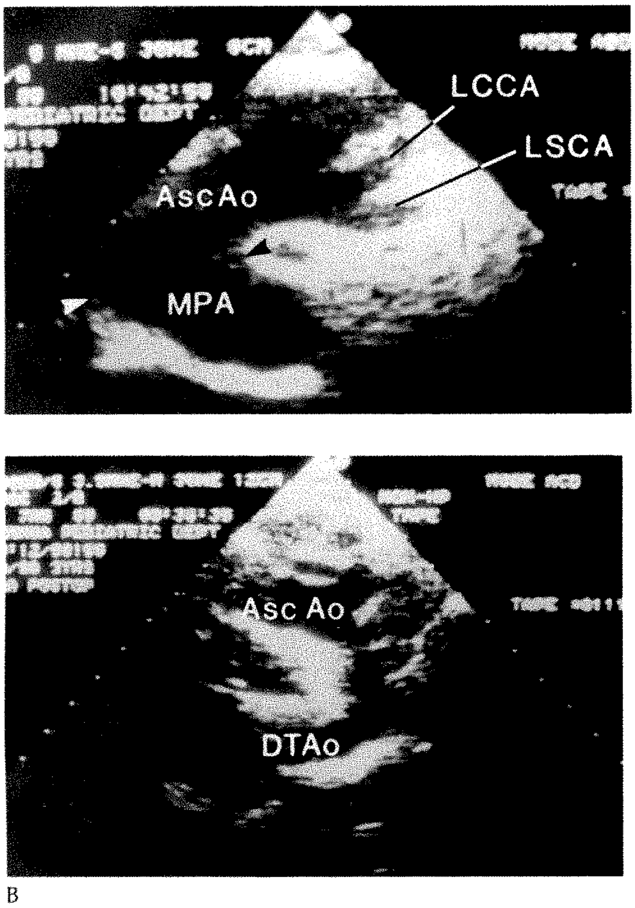

Fig 1. (A) Preoperative echocardiogram (suprasternal long-axis view) showing type A interrupted aortic arch beyond the left subclavian artery (LSCA). *The absence of the aortopulmonary septum (huge aortopulmonary window) between the ascending aorta* (AscAo) *and main pulmonary artery* (MPA) *is indicated by the* arrowheads. *(B) Postoperative echocardiogram (suprasternal long-axis view) 3 months after operation showing the aortic arch now in continuity after direct anastomosis to the descending thoracic aorta* (DTAo). (LCCA *= left common carotid artery.)*

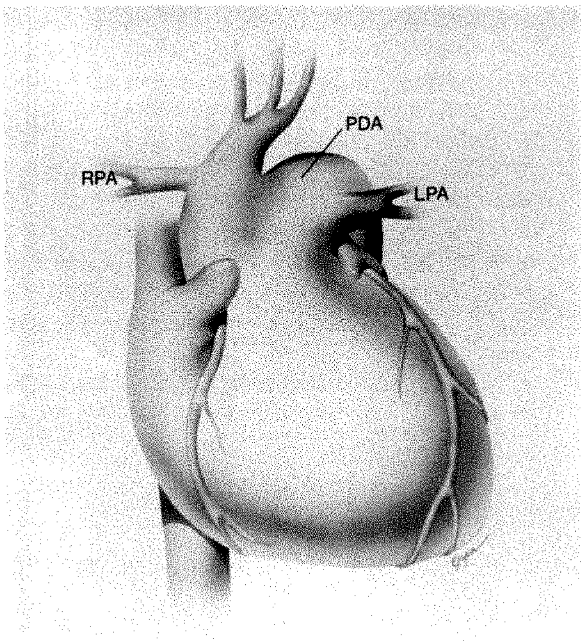

Fig 2. External appearance of the heart is similar to that of type III truncus arteriosus with interrupted aortic arch. The right pulmonary artery (RPA) *arises from the right side of the ascending aorta.* (LPA *= left pulmonary artery;* PDA *= patent ductus arteriosus.)*

controlled with a clamp and the left pulmonary artery, with digital pressure. When the heart fibrillated at 29°C, the distal ascending aorta was clamped. The ascending aorta was opened transversely at the level of the right pulmonary artery and cardioplegic solution was infused directly into the coronary ostia. There was almost complete absence of the aortopulmonary septum with just a 5-mm septum between the two semilunar valve annuli. The diameter of the defect was 25 mm, whereas the right pulmonary artery origin was 12 mm in diameter. An extensive baffle was fashioned from a 12-mm collagen-impregnated, knitted Dacron tube graft that was opened longitudinally. At its left end the graft was opened out and sutured to the circumference of the large aortopulmonary defect with particular care taken in the region of the left coronary ostium. Posteriorly the graft was fashioned to create a tunnel directing pulmonary arterial blood to the ostium of the right pulmonary artery (Fig 3). The aorta was closed by direct suture. After clamp release and rewarming to normothermia, the child was separated from bypass without difficulty. A pulmonary artery line was inserted through the infundibulum of the right ventricle into the left pulmonary artery. In the operating room pulmonary artery pressure was approximately half of the systemic pressure.

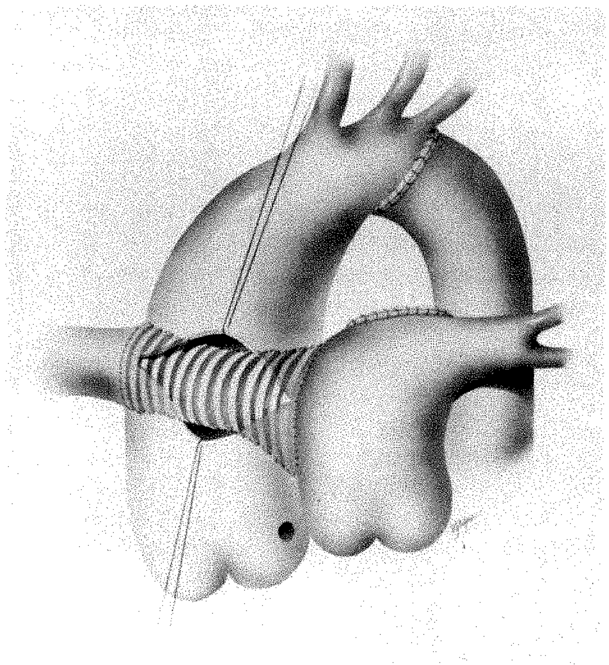

Fig 3. The interrupted aortic arch has been repaired by direct anastomosis. A Dacron baffle directs pulmonary arterial blood from the aortopulmonary window to the right pulmonary artery.

The child was extubated on the first postoperative morning. There was no step-up in oxygen saturation between the right atrium and pulmonary artery. Pulmonary artery pressure was one third of the systemic pressure, as was right ventricular pressure on pullback of the line. There was a 20-mm Hg gradient between right arm and leg blood pressures.

The child was discharged on the 14th postoperative day and continues to do well clinically 8 months postoperatively. Two-dimensional echocardiographic examination 3 months postoperatively (Fig 1B) revealed no gradient in the supraaortic region or between the main pulmonary artery and right pulmonary artery. There was a 25 to 30-mm Hg gradient between the aortic arch and descending aorta.

Comment

Probably the most intriguing aspect of this case is the particularly low pulmonary artery pressure that was recorded postoperatively. This was in spite of the absence of severe symptoms of congestive heart failure preoperatively in this nearly 3-year-old child. The child had never been treated with diuretic agents and had only moderate cardiomegaly preoperatively. Nevertheless, pulmonary artery pressure decreased from half of the systemic pressure immediately after discontinuation of cardiopulmonary bypass to one third of the systemic pressure 24 hours postoperatively.

In 1974, Daily and colleagues [3] reported the repair of a similar constellation of anomalies in a 16-year-old child. However, that child died 2 years after operation with evidence by catheterization and histology of pulmonary vascular disease. Unfortunately, a lung biopsy was not performed in our patient.

The surgical techniques that were described in the two previous case reports of this anomaly probably could not have been applied successfully to our patient. The technique described by Berry and associates [2] of reimplantation of the right pulmonary artery in the main pulmonary artery would have presented the problem of a large size discrepancy between the right pulmonary artery (12 mm) and the diameter of the aortopulmonary defect (25 mm). The patient of Berry and associates was a neonate in whom elasticity and mobility of vessels is greater. The technique of Daily and colleagues [3] of a complete tube graft from the aortic annulus to the internal orifice of the proximal arch of the aorta would have also presented a problem of size disparity. The technique is also less suitable in a younger growing child relative to their patient who was 16 years of age.

Finally, this report illustrates that in spite of the setbacks of the Cultural Revolution, a spirit of close cooperation between Chinese and American surgical teams has in a short space of time enabled the repair of even the most complex congenital heart lesions within the People's Republic of China.

We thank Project Hope, particularly John Walsh and William Walsh, Jr, for their efforts in coordinating our collaborative education program between Xin Hua Hospital, Boston Children's Hospital, Boston, and Children's Hospital of Philadelphia, Philadelphia.

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Echocardiographic Diagnosis and Surgical Treatment of Postpartum Pulmonary Embolism

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Pulmonary embolism remains a problem in the United States in terms of both morbidity and mortality. New diagnostic modalities to make rapid diagnosis are now available, and allow for bedside diagnosis of pulmonary embolism without the use of pulmonary angiography. As a reference, a case involving a postpartum patient is reviewed. Use of echocardiography, a device readily available even in small institutions, allowed for early diagnosis and institution of therapy in this particular case and in others. Diagnostic features of pulmonary embolism are discussed and the literature is reviewed.

(Ann Thorac Surg 1990;49:667–9)

Pulmonary embolism remains a major cause of morbidity and mortality in the United States, with an estimated 500,000 nonfatal pulmonary emboli each year, and is responsible for or contributes to the death of 142,000 people each year [1].

To date, diagnosing pulmonary embolus has been a time-consuming process. In a patient in stable condition, noninvasive radioisotope scans have been employed; for patients in an unstable condition believed to have an embolus, pulmonary angiography has been used before implementation of therapy. A substantial portion of these latter patients have died.

A 28-year-old woman was admitted for elective cesarian section. Physical examination was unremarkable and laboratory studies were normal. She underwent cesarian section and a simultaneous tubal ligation was performed.

Twelve hours later she was reported to have a distended abdomen with hematuria and hematocrit of 0.24. Despite fluid administration, her blood pressure remained low, her pulse was elevated to 140 beats/min, and her respiratory rate was 40 per minute. She was brought to the operating room for exploration. At laparotomy, approximately 3,000 cm^3 of clotted blood was found in her abdominal cavity. A tear in the left ovarian vein was identified and ligated. A left salpingo-oophorectomy was performed.

Intraoperatively, she had labile blood pressure, tachycardia, a new right bundle-branch block, and what was subsequently identified as paroxysmal ventricular tachycardia. Shortly after her return to the recovery room, the patient was hypotensive and tachycardic, and had a persistent right bundle-branch block. Her arterial oxygen tension on 90% oxygen and positive end-expiratory pressure of 10 cm H_2O was only 131 mm Hg. She had persistent ventricular ectopy. A central venous pressure recording was performed; central venous pressure was found to be 26 cm H_2O. Chest roentgenogram (Fig 1) showed a dilated cardiac silhouette.

Echocardiography showed a large right ventricle and distended right atrium in addition to a distended inferior vena cava. The left ventricle was compressed with a flat intraventricular septum, which exhibited neutral or paradoxical motion during systole; the left atrium was also small and compressed. A large serpiginous highly mobile echocardiographic density was imaged in the right atrium, traversing the tricuspid valve and exhibiting vigorous motion (Fig 2). The configuration was most consistent with a clot that had migrated from one of the large veins. During the course of the test, the leading edge of the "clot" was seen to migrate through the outflow tract of the right ventricle across the pulmonary valve and into the main pulmonary artery. This clot was imaged best in a standard short-access view as well as in the foreshortened apical (inflow, outflow) view. A separate clot was seen in the right pulmonary artery close to the bifurcation through the suprasternal window (Fig 3). There was severe tricuspid insufficiency seen by color Doppler echocardiography.

The patient was emergently brought to the operating room. Pulmonary embolectomy was performed through a standard median sternotomy incision on total cardiopulmonary bypass with bicaval cannulation. The right heart was massively dilated. After atriotomy, no clot was seen in the right atrium or right ventricle. Pulmonary arteriotomy was performed and revealed massive clot in the artery, which was removed with suction, irrigation, and forceps. The right main pulmonary artery was then opened just proximal to its exit from the pericardium and additional clot was removed. No clot could be extruded by applying pressure from the lung proximally. Initial attempts to wean from bypass were unsuccessful because of very high right-sided pressures. The patient was subsequently weaned off bypass but required inotropic support. A Greenfield vena caval filter was then placed through a cervical approach for prophylaxis. The patient

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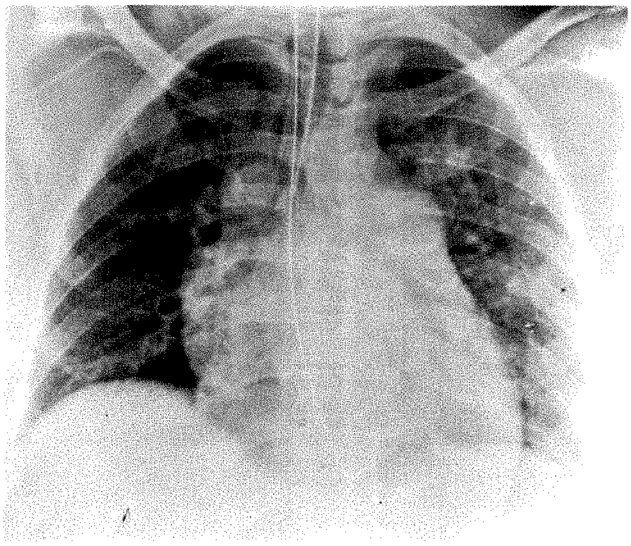

Fig 1. Portable chest roentgenogram showing dilated heart.

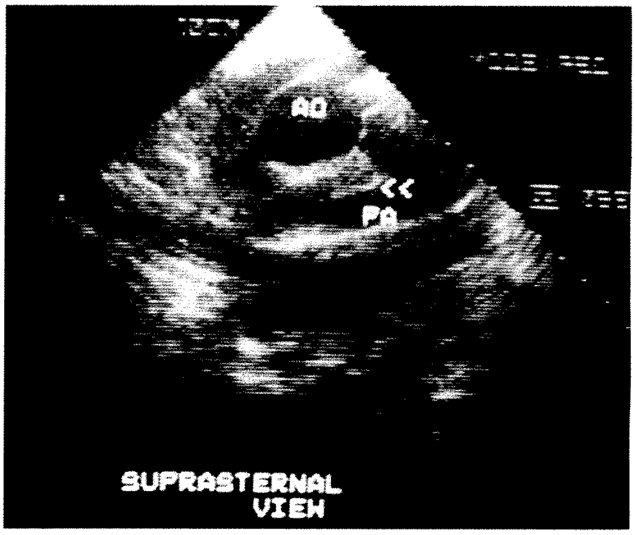

Fig 3. Echocardiogram (suprasternal view) showing clot in main pulmonary artery (arrow). *(*AO = *aorta;* PA = *pulmonary artery.)*

was extubated six hours postoperatively and remained hemodynamically stable. She had an uneventful postoperative course and went home on the 15th postoperative day. Follow-up ventilation/perfusion scans showed no perfusion abnormalities (Fig 4).

Comment

Although the therapy of massive pulmonary embolus necessitates a major surgical procedure, there is no alternative that offers a good chance of survival. Most importantly, the diagnosis of pulmonary embolism must be made early and therapy implemented. This is facilitated by a high index of suspicion in a gravely ill patient. In the case we report, echocardiography was used as the sole diagnostic modality before surgical intervention.

The diagnosis of pulmonary embolus is commonly made by the indirect method of ventilation/perfusion nuclear scans. The "gold standard" for the diagnosis of pulmonary embolus remains the pulmonary angiogram. There have been several reported cases [2, 3] of pulmonary emboli diagnosed by two-dimensional echocardiography with use of the suprasternal approach. With this technique, pulmonary emboli are rapidly diagnosed and directly imaged. In the case we report here, we observed both intracardiac clot and clot in the pulmonary artery. There was no clot found in the heart after cardiotomy, suggesting that it had embolized. In serial studies performed while awaiting transfer of this patient, the clot was seen to migrate into the right ventricular outflow tract.

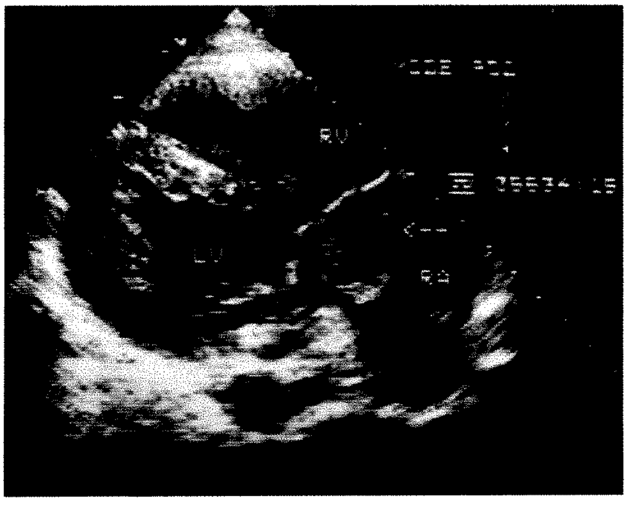

Fig 2. Echocardiogram of clot in right atrium (arrow *shows leading edge). (*LV = *left ventricle;* RA = *right atrium;* RV = *right ventricle.)*

The use of echocardiography in the diagnosis of intracardiac thrombus is well documented. In one case report [4], a two-dimensional echocardiogram showed a clot moving back and forth across the tricuspid valve, similar to that of the case we report here. However, that patient was treated with streptokinase after cardiac catheterization and died 30 hours after the start of thrombolytic therapy.

The antemortem diagnosis of right-sided cardiac emboli is rare. As of July 1988, only 53 patients had been reported in the English-language literature with a mass in the right heart demonstrated by echocardiography and subsequently definitively diagnosed as pulmonary embolus [5–7]. In 5 of these patients, a mass was seen flipping back and forth across the tricuspid valve. Of 33 patients treated medically, half died, whereas only 15% of surgically treated patients died [8].

The echocardiographic appearance of right atrial masses has been described as elongated, fusiform in character, lacking in discrete point of attachment, and often free-floating in the right side of the heart [7]. The acoustic density is less than that of normal myocardium and distinct from that of myxomas. This description is in contrast to that for left-sided thrombi. Other right-sided masses that may be confused with thromboemboli include

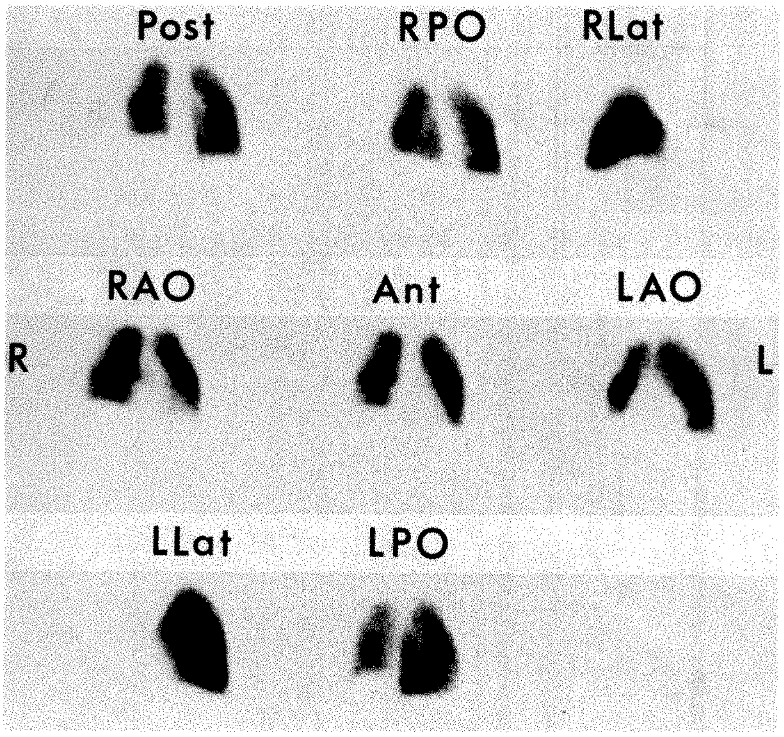

Fig 4. Nuclear ventriculography 4 months after operation revealing normal ventilation and perfusion scans. (Ant = *anterior;* LAO = *left anterior oblique;* LLat = *left lateral;* LPO = *left posterior oblique;* Post = *posterior;* RAO = *right anterior oblique;* RLat = *right lateral;* RPO = *right posterior oblique.)*

myxomas and vegetations, which can usually be differentiated on the basis of the characteristics noted previously. The use of echocardiography is expanding in the diagnosis of thromboembolism because many patients do not show symptoms suggestive of emboli, and the echocardiogram is often the initial examination. Echocardiography is a rapidly applied technique that appears to give good results in the diagnosis of thromboemboli in the right heart, as well as emboli in the proximal pulmonary artery.

In patients who become acutely unstable, the index of suspicion must be high for the diagnosis of pulmonary embolism. Diagnosis can then be confirmed by pulmonary angiography or echocardiography. We recommend echocardiography as a rapid method of diagnosing intracardiac or pulmonary artery emboli. In addition, evidence of right heart volume and pressure overload can easily be obtained. If the patient has tricuspid insufficiency, pulmonary artery pressures can be estimated noninvasively. Once the diagnosis is confirmed, rapid transportation to the operating room and pulmonary embolectomy under cardiopulmonary bypass is recommended to maximize survival. Vena caval interruption with a clip or filter is essential to prevent recurrence.

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Traumatic Intercostal Pulmonary Hernia

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Traumatic intercostal pulmonary hernia is a rare sequel to injury, especially in children and where there is no penetrating wound. Spontaneous regression of these hernias has been reported and conservative management has been advocated. We describe a case in which a smooth-walled intercostal defect and lung hernia that did not regress developed in a child. This was successfully repaired with a Gore-Tex (expanded polytetrafluoroethylene) patch. Repair is advisable in such cases, but operation can be delayed to allow possible resolution.

(Ann Thorac Surg 1990;49:670–1)

An active 13-year-old boy injured his right side on the handlebars of his bicycle. Shortly after, he noticed a plum-sized lump appear on his chest wall at the site of injury that was present only on forced expiration against a closed glottis (Fig 1). The lesion slowly increased in size. The child had previously been fit apart from childhood asthma.

When he was admitted to the hospital 6 months later, no abnormality could be seen during normal breathing, but a clearly defined defect about 5 cm long was detected between the sixth and seventh ribs in the anterior axillary line. On performing a Valsalva maneuver, a fluctuant swelling appeared at this site.

Chest roentgenogram revealed no skeletal abnormality, but a film taken during a Valsalva maneuver demonstrated air within the lung lying in a plane outside the ribs. A diagnosis of acquired lung hernia was made and surgical repair was deemed advisable.

Repair was performed under general anesthesia and with muscle relaxation. An incision was made over the defect in the intercostal muscles and in the line of the ribs. A clearly defined and smooth-edged breach in the intercostal muscles was found and an easily identified pleural sac was seen beneath it. The sac was opened, there being no adhesions between this and the underlying lung (Fig 2).

It was clear that the size of the hole (5 cm × 2 cm) and the fixity of the intercostal muscles and parietal pleura precluded direct closure. A small patch of Gore-Tex (expanded polytetrafluoroethylene; W.L. Gore, Dunfirmline, Scotland) was therefore fashioned and sewn into the intrathoracic side of the defect with a continuous running 4-0 Prolene (Ethicon, Edinburgh, Scotland) suture. The fascia and skin were then closed with Vicryl sutures.

The boy made an uneventful postoperative recovery, no residual chest-wall defect being demonstrable.

Comment

Lung hernias were first classified by Morel-Lavelle [1]. A summary of this classification is given in Table 1 and the added frequencies show that about half of lung hernias are a consequence of trauma [2]. However, the overall incidence is low even in times of war and the condition is especially uncommon in children. (We have identified 5 previously reported cases [3–7].)

Although blunt trauma is known to cause these hernias, they more usually follow a penetrating injury. The appearance of the hernia is often delayed by several weeks (where they are called "consecutive," but might be better classified "delayed") or even up to 40 years [4]. The hernia is usually formed at the site of trauma (in the case of penetrating injury) or anteriorly or posteriorly where the intercostal muscles exist as a single internal or external layer only (blunt trauma) [8].

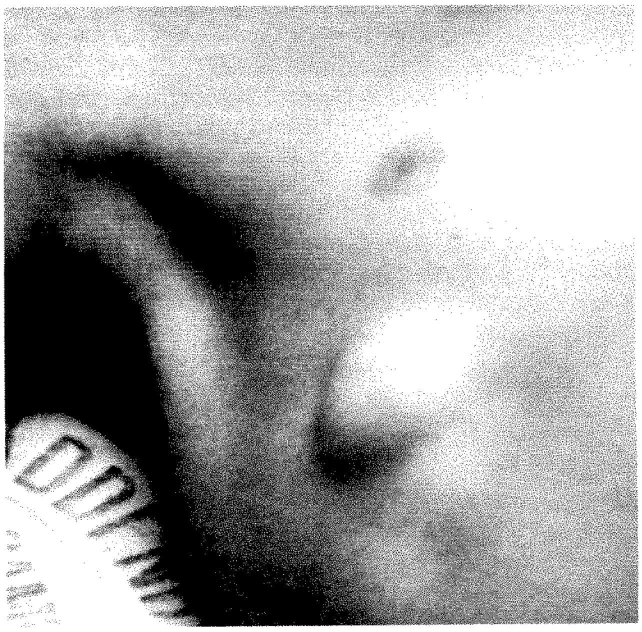

Fig 1. The intercostal hernia during a Valsalva maneuver.

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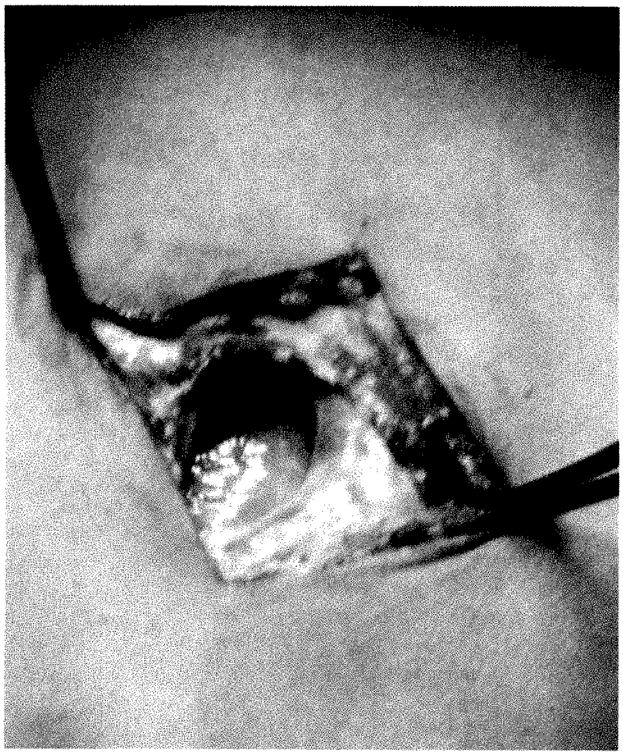

Fig 2. The defect at operation showing normal lung beneath the breach in the intercostal muscles.

In patients with blunt trauma who are explored the defect is often described as a muscular tear [5]. This suggests a mechanism in which the ribs deform excessively without fracture (as they are known to do in children), the force thus being transferred to the weaker, single-layered intercostal muscle, which tears.

Table 1. Classification of Lung Hernias (After Morel-Lavelle [1]) With Relative Frequencies (Hiscoe and Digman [2])

Type	Frequency
Congenital: rib or intercostal hypoplasia or agenesis	18%
Aquired	
Traumatic including consecutive	52%
Pathological or spontaneous	30%

Some controversy exists concerning the place of surgical repair. Some authorities advocate operation for these hernias as a matter of routine [9], but the observation that spontaneous regression can occur has led to support for conservative management, and some reports describe the beneficial effects of strapping the hernia site [5, 6]. However, in none of these latter cases is there mention of a palpable intercostal defect. In the case described in this report, not only was there an easily palpated and relatively large defect, but also no regression had occurred 6 months after the injury. Thus we believed operation to be indicated.

At operation the size of the defect and the fixity of its margins led us to repair the hernia with a synthetic patch (we used expanded polytetrafluoroethylene, but successful use of other sheets and meshes has been described and a fascia lata graft might be an ideal material). However, it has been suggested that all defects can be closed with a combination of plastic procedures using chest wall structures: rib, periostal flaps, and muscle.

In conclusion, we suggest that because traumatic intercostal hernias are rarely symptomatic or complicated, it is reasonable to delay intervention for a period to allow spontaneous resolution. However, if this does not occur, or if a large or sharply defined defect is found, surgical repair should be undertaken. The defect may be repaired by a local plastic procedure, with a synthetic patch, or with a fascia lata graft.

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Multiple Congenital Aneurysms of the Atria

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Aneurysms of both the left and right atria are very rare congenital abnormalities. A 3-year-old boy underwent operation with preoperative diagnosis of aneurysms of both atria and supraventricular tachyarrhythmia. The multiple aneurysms were resected during cardiopulmonary bypass. The patient was asymptomatic after operation and has maintained sinus rhythm.

(Ann Thorac Surg 1990;49:672–3)

Congenital aneurysms of the atria are very rare anomalies. The lesions are usually associated with supraventricular arrhythmias and thromboembolism. We describe a patient who had multiple aneurysms in both atria.

A 3-year-old boy who had been diagnosed to have supraventricular tachyarrhythmia during his fetal life was referred to our institution for further evaluation and management. On admission, the patient was not in acute distress and showed normal development. His body weight was 10.4 kg and there was no sign of heart failure. The electrocardiogram revealed supraventricular tachycardia with a fast ventricular response of 167 beats/min. A chest roentgenogram showed mild cardiomegaly. The patient was digitalized. In cross-sectional echocardiography, there was a narrow, long echo-free space behind and lateral to the left ventricle. The space appeared to be continuous with the left atrium (Fig 1). Cardiac catheterization revealed normal intracardiac pressure, no evidence of mitral valve disease, and no intracardiac shunt. Injection of contrast material in the superior vena cava indicated a "hump" in the right atrium (Fig 2, left) and a large aneurysm connected to the left atrial appendage (Fig 2, right). Resection of the aneurysms was recommended to obviate the need for long-term digitalization and anticoagulants.

At operation through a median sternotomy, multiple aneurysms in both right and left appendages were discovered (Fig 3). The pericardium was intact. Extracorporeal circulation was instituted and myocardial protection was achieved with cold cardioplegia. The aneurysms were resected and the defects were sutured directly. The tricuspid and mitral valves were entirely normal. There were no thrombi in the aneurysms. The biggest aneurysm measured 3 cm. Histological examination of the wall of the aneurysms revealed that they were composed of fibrous tissue and attenuated myocardial fibers with an endothelial lining.

The postoperative course was uneventful, and the patient was discharged free of symptoms and in sinus rhythm.

Comment

Congenital atrial aneurysm is a rare entity. Congenital isolated left atrial aneurysm was first described in 1953 by Fry [1]. There have been more than 30 cases reported [1–8]. However, we could find only two reports of congenital right atrial aneurysm [4, 8]. Therefore, multiple aneurysms of both atria is considered to be an extremely rare anomaly. A review of the literature indicates that ours is the second report of congenital intrapericardial aneurysms of both atria [7]. Although the etiological process is not clear, one of the possibilities is that a congenitally weak portion of the atrial wall progressively dilates [3, 7].

Of the reported cases of left atrial intrapericardial aneurysm [1–8], 75% of the patients showed symptoms of supraventricular arrhythmia and were treated with digitalis. A few patients had heart failure. In less than half of the patients, systemic thromboembolism was part of the

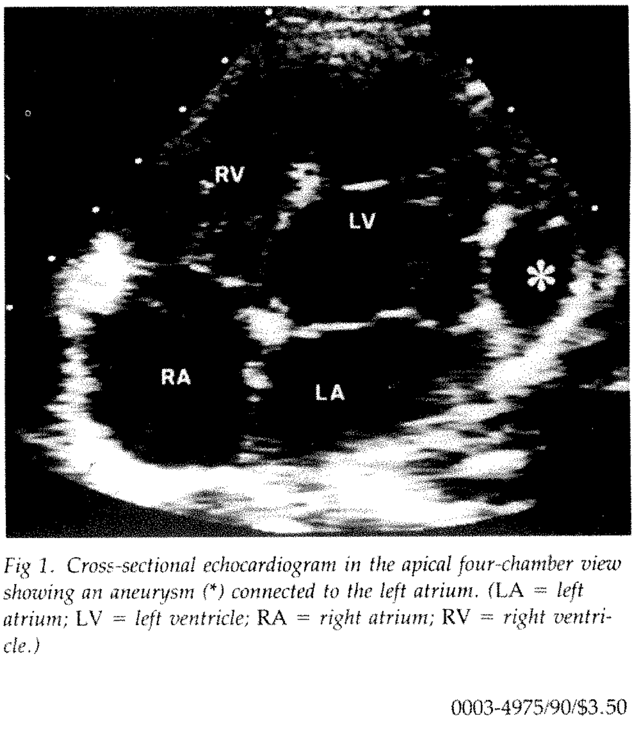

Fig 1. Cross-sectional echocardiogram in the apical four-chamber view showing an aneurysm (*) connected to the left atrium. (LA = *left atrium;* LV = *left ventricle;* RA = *right atrium;* RV = *right ventricle.)*

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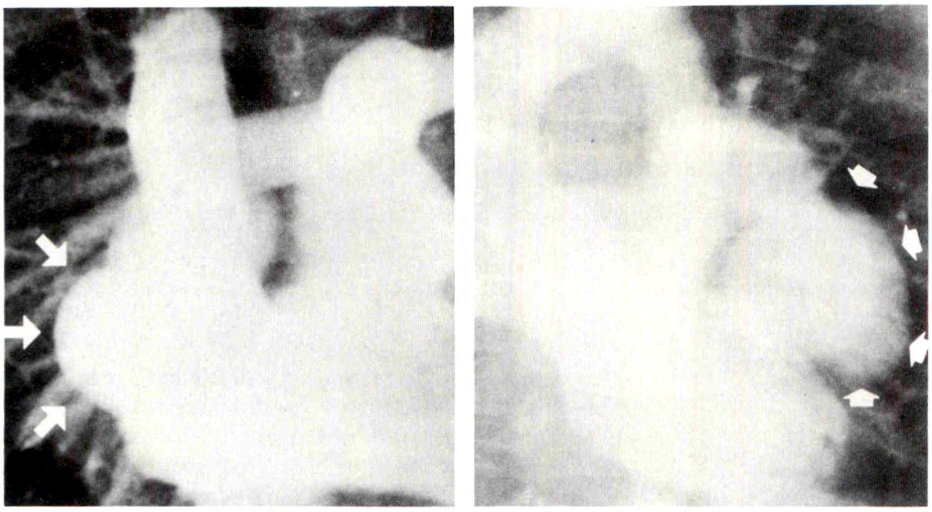

Fig 2. (Left) Angiogram with injection in superior vena cava. A hump (arrows) *is seen in the right atrium. (Right) A big aneurysm* (arrows) *continuous with the left atrial appendage is shown in levophase.*

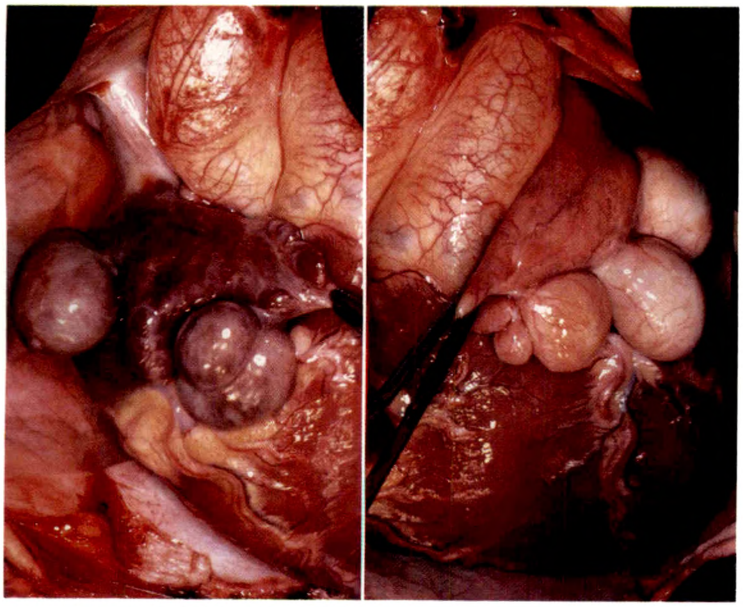

Fig 3. Operative view of the aneurysms in the right (left) and the left appendages (right).

initial symptoms. The diagnosis of congenital atrial aneurysm has been suggested by cardiomegaly, abnormal electrocardiogram, cross-sectional echocardiography, and radionuclide angiography [2, 5]. Angiocardiography, however, has been the definitive method of establishing the diagnosis of aneurysm [3]. Our patient was diagnosed to have supraventricular tachycardia by fetal electrocardiogram during his intrauterine life.

Surgical excision is recommended to eliminate a potential source of emboli and for relief of supraventricular arrhythmia. Operation is an effective therapeutic measure. Seventy-five percent of patients were reported to maintain sinus rhythm postoperatively. No patients have died as a result of surgical intervention, nor have any perioperative emboli been reported [1–8]. Although most lesions can be excised without cardiopulmonary bypass and through thoracotomy, cardiopulmonary bypass with cardioplegic arrest is considered to provide additional protection from intraoperative embolization [3, 6].

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Venous Cannulation for High-Flow Femorofemoral Bypass

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A simple technique is described for reliable passage of a large-diameter venous cannula from a femoral venotomy into the right atrium, to facilitate high-flow femorofemoral bypass.

(Ann Thorac Surg 1990;49:674–5)

Cannulation of the femoral artery and vein allows rapid institution of cardiopulmonary bypass; however, the high flow rates necessary for complete decompression of the heart, or for rapid cooling and rewarming, cannot be achieved unless a suitably large venous cannula can be positioned with its tip in the right atrium [1, 2]. It is often difficult to pass a cannula through the concavity of the pelvis, past lumbar veins and the caval bifurcation, especially if the patient is in decubitus position. A method is described that allows rapid, reproducible right atrial cannulation with any size cannula capable of entering the common femoral vein.

Material and Methods

The femoral vessels are exposed and encircled with tapes. The patient is heparinized. A guidewire (180-cm Rosen wire, 0.089 cm [0.035 in.] diameter; TCSM-35-180-1.5 Rosen, Cook, Bloomington, IL) is inserted with Seldinger technique and passed into the right atrium. Position is best confirmed by transesophageal echocardiography, but can also be confirmed by fluoroscopy or (with less certainty) by estimation based on premeasurement of the approximate distance from the groin to the atrium. A cannula (Argyle chest tube) with an external diameter slightly larger than the vein is selected. A chest tube is used because of its length (51 cm) and its end-hole/multiple-side-hole configuration. A limited number of cannulas designed for venous drainage and offering similar advantages are available commercially.

The cannula (chest tube) is prepared for insertion by placing a large angioplasty balloon catheter (7F, 75-cm catheter; balloon length, 4 cm; balloon diameter, 10 mm; DC/10-4/7/75 Medi-Tech, Watertown, MA) in the lumen

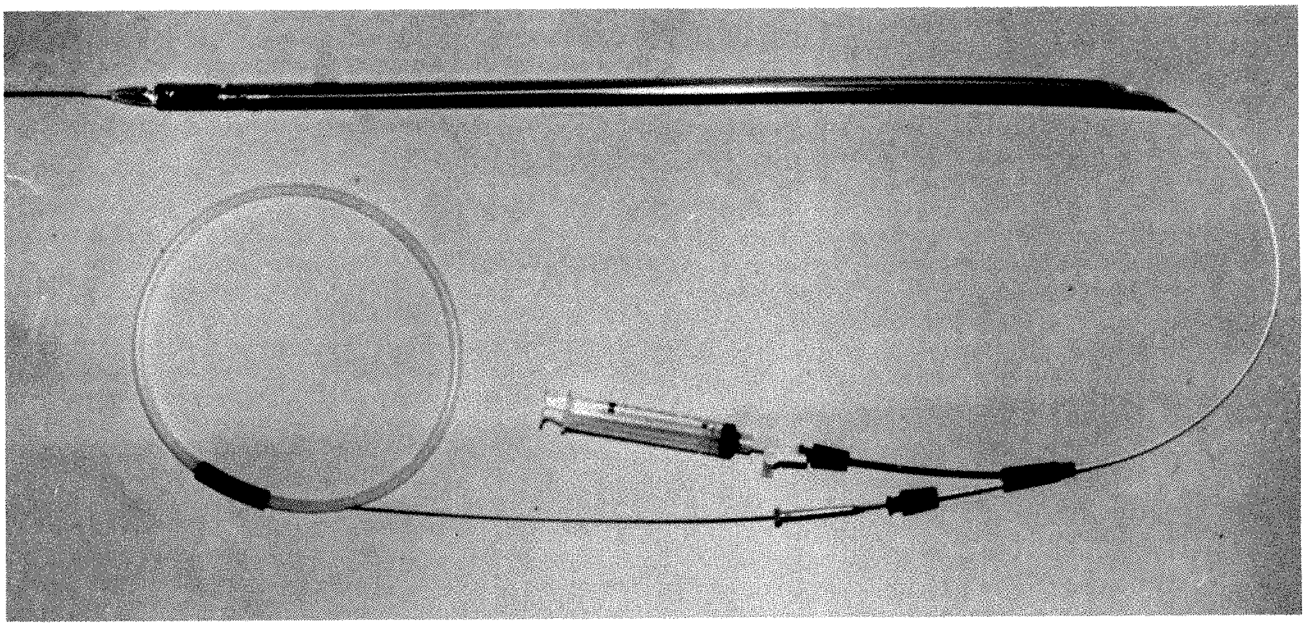

Fig 1. A No. 36 Argyle chest tube is shown with an angioplasty balloon catheter inflated at its tip and with the central lumen of the angioplasty catheter-chest tube assembly threaded over a guidewire.

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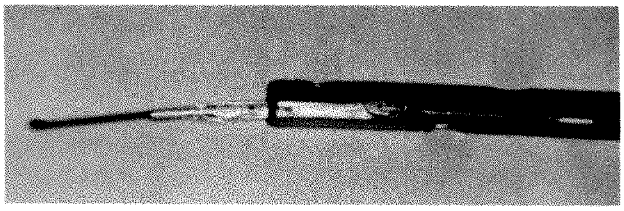

Fig 2. The angioplasty balloon catheter is shown, deflated and threaded over a guidewire, positioned where it will be inflated at the tip of the chest tube.

(Fig 1) with half the balloon extending beyond the cannula tip (Fig 2). The balloon is inflated, tightly wedging the balloon in position, converting the blunt tip of the cannula into a smoothly tapering nosecone with a central lumen that will follow the guidewire (Fig 3).

The femoral vein is clamped proximally and distally, and a transverse venotomy is made at the site of entry of the guidewire. The central lumen of the angioplasty balloon catheter is threaded over the guidewire, then the cannula assembly is passed into the vein over the guidewire as the proximal clamp is released. With gentle pressure the cannula will follow the guidewire into the right atrium. Blood loss can be minimized by applying a tube clamp to the entire assembly, which does not prevent balloon deflation. Once correct position is confirmed, the balloon is deflated, the tube clamp is released to allow quick withdrawal of the catheter-guidewire assembly, and the cannula is connected directly to the venous line of the bypass circuit. Cannulas of 36F to 40F can be inserted in most adults with this technique, and flows nearly equivalent to direct atrial cannulation can be achieved.

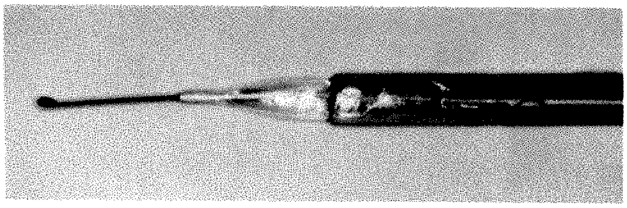

Fig 3. The angioplasty balloon catheter, threaded over a guidewire, is shown fully inflated with about half the balloon lying distal to the tip of the chest tube. The balloon is tightly wedged in position, and imparts a smoothly tapering configuration to the leading end of the tube.

Clinical Example

This technique was first used in a 78-kg, 27-year-old man with a giant intracerebral aneurysm. A No. 22 Bardic straight arterial cannula was placed in the right common femoral artery, and a No. 36 Argyle chest tube was placed in the right atrium using the method described. Position in the right atrium was confirmed with intraoperative transesophageal echocardiography. Flows of 3.8 to 5.0 L/min were obtained, and an esophageal temperature of 15°C was achieved in 52 minutes, followed by 38 minutes of circulatory arrest and 95 minutes of rewarming. It could be seen by transesophageal echocardiography that the heart emptied promptly on bypass and remained empty throughout, whether beating or fibrillating. The arteriotomy and venotomy were closed routinely without sequelae.

Comment

Femorofemoral bypass is an important technique for: (1) rapid institution of bypass outside the operating room, (2) control of the circulation while obtaining initial exposure in a difficult reoperation, (3) decompression of the right heart during single-lung transplantation in patients unable to tolerate pulmonary artery occlusion, and (4) management of profound hypothermia and total circulatory arrest in descending thoracic aortic procedures and neurosurgical procedures. High flow rates are necessary to completely decompress the heart or to allow rapid cooling and rewarming, and a centrally placed, large-diameter venous cannula is a prerequisite for high-flow femorofemoral bypass in perfusion systems based on gravity drainage of venous blood [1, 2].

The simple technique described allows reliable introduction of the largest possible venous cannula into the right atrium through a femoral venotomy. In retrospect, review of the literature reveals that this method is a vascular application of a technique for percutaneous insertion of large, untapered catheters into abscess cavities originally described by Kerlan and associates [3].

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Forceps Extraction of Permanent Pacing Leads

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With the advent of tined transvenous cardiac pacing leads, the complete extraction of pacing leads in the treatment of an infected cardiac pacing system has become increasingly difficult. A method is described for the extraction of permanent pacing leads from the heart using alligator forceps inserted transvenously through the right internal jugular vein, grasping the lead near its insertion point in the cardiac muscle.

(Ann Thorac Surg 1990;49:676–7)

Infection of a permanent pacing system after implantation remains an uncommon but serious problem. Many techniques for the treatment of infected pacing systems have been described. On some occasions, simple opening of the wound, debridement, and closure over antibiotic irrigation catheters have been successful. In chronically implanted systems, success has been obtained by removal of the pulse generator and division of the lead before its entry into the infected pocket with retention of the cardiac portion of the lead. In patients in whom the lead is obviously involved with infection or in cases of recurring infection on a retained lead, complete removal of all foreign bodies associated with the pacing system is necessary for eradication of the infection. In a system implanted for a short time, the lead can usually be easily removed with mild traction. However, in systems implanted for a long time, the lead becomes encased in a fiber sheath preventing easy extraction by traction.

Although this problem occasionally occurred in previous years with simple ball tip leads, the advent of tined fixation devices on the end of the leads has lead to far more frequent problems in the complete extraction of these leads. Traction frequently leads to breakage of the insulating cover, uncoiling of the conductor, and ultimate breaking of the lead with portions of the lead in varied amounts remaining in the heart and the venous system. Previously described techniques for the removal of these embedded leads include constant traction with weight and pulley devices [1], loop snare devices using angiographic techniques [2], thoracotomy with limited cardiotomy [3, 4], dotter [5] or Dormier [6] basket extraction, and special dilator extractor devices [7, 8]. All these techniques have been useful and should be included in the surgeon's armamentarium when approaching these difficult cases. We have evolved a method of lead extraction using alligator forceps that has been dependable and safe for the complete removal of these leads when other methods have been unsuccessful.

Technique

The patient is positioned in the supine position on the operating table with the head turned to the left. The entire neck, chest, and upper abdomen are prepared to provide adequate exposure for approach to the right internal jugular vein or the previous pacemaker pocket and median sternotomy incision if necessary. If not already accomplished, the pacemaker pocket is opened, the pulse generator is removed, and the permanent lead is removed by general traction if possible. In cases in which the permanent lead cannot be removed by traction or the lead has been broken by previous attempts to remove it, a jugular approach is undertaken. The jugular vein is entered using an anterior or posterior approach, and a guidewire is passed into the internal jugular vein and down the superior vena cava. Over this guidewire, a dilator and sheath are passed. The size of the sheath is selected as is appropriate for the size of the grasping forceps to be used. Usually 12F or 14F sheaths are used. The tear-away split sheaths are favored so that once the grasping forceps are placed, the sheath may be removed, eliminating the chance of air embolism. Once the sheath is placed in the superior vena cava, rigid sterile grasping forceps are passed through the sheath and into the superior vena cava. Grasping alligator forceps such as used for rigid bronchoscopy are suitable for this task. The grasping forceps are passed carefully under fluoroscopic observation until they are quite close to the tip of the pacemaker wire (Fig 1). It is usually not necessary to cross the tricuspid valve to remove the pacemaker lead. The lead is grasped, and firm steady traction using the grasping forceps is used to remove the entire lead system through the percutaneous venotomy in the neck.

Although we have not experienced perforation of the heart using this technique, forceful traction on pacemaker leads for removal has been reported to lead to pericardial bleeding and, therefore, this procedure should never be performed without the patient prepared in the operating room for immediate sternotomy should pericardial tamponade develop.

We have performed this procedure on 4 patients. Two patients had ventricular leads entrapped in the right ventricular chamber. The leads had been broken during previous removal attempts using traction. The remnant of the lead was easily grasped and removed. A third patient had an atrial lead screwed into the lateral wall of the right atrium. This lead could not be unscrewed and removed using the usual methods; however, by grasping the lead near the tip and rotating the grasping forceps, we were

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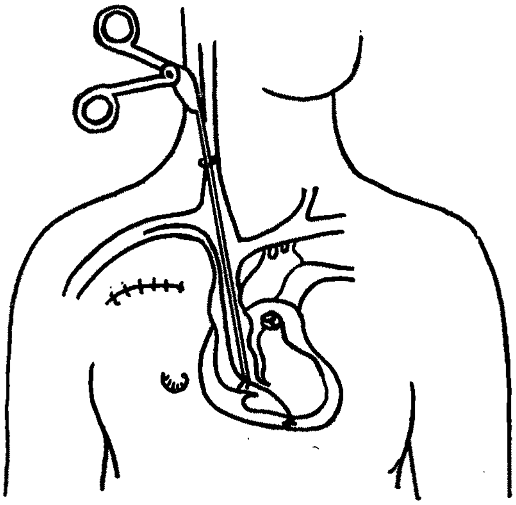

Fig 1. Removal of the chronic pacing lead by grasping forceps applied near the distal end of the lead.

able to unscrew the lead and to remove it easily from the right atrial wall. In the fourth patient, we were able to dislodge a lead from the right ventricular chamber using traction and pull it nearly out of the body; however, the lead broke as the tip and approximately 7½ cm of the lead approached the subclavian superior vena cava junction. We then passed a grasping forceps through the internal jugular vein, grasped the lead, and removed it completely.

We believe this method, although not frequently required, is an occasionally useful tool and is preferable to performing a thoracotomy and cardiotomy for the removal of a permanently entrapped pacing lead.

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Notice From the American Board of Thoracic Surgery

The part 1 (written) examination will be held at the Hyatt-Regency, Dallas Fort Worth Airport, Dallas TX, on February 3, 1991. The closing date for registration is August 1, 1990.

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CLASSICS IN THORACIC SURGERY

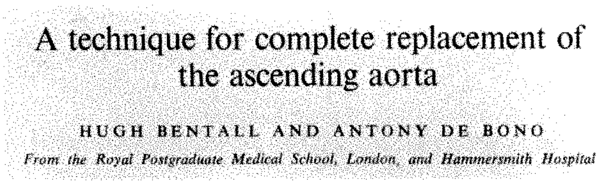
Toward a Durable and Safe Aortic Root Reconstruction

Hovald K. Helseth, MD

Department of Surgery, Hennepin County Medical Center, Minneapolis, Minnesota

The development of acceptable and durable aortic root operations has been interesting. With trial and error there has been a progressive intrusion on the aortic valve and coronary arteries. Many surgeons have contributed to this progression, with the final step being taken by Dr Bentall in 1968. Since that time has come a gradual application of this more complete operation and a better understanding of its advantages.

(Ann Thorac Surg 1990;49:678–9)



A technique for complete replacement of the ascending aorta

HUGH BENTALL AND ANTONY DE BONO

From the Royal Postgraduate Medical School, London, and Hammersmith Hospital

Fig 1. Title page of the initial report by Bentall and De Bono. (Reprinted from [5] by permission of the British Medical Association.)

In contrast to the grandiose presentation of many surgical techniques stands the succinct case report of Drs Bentall and De Bono in *Thorax* in 1968. The former entice and fade away; however, this brief description of a single patient encouraged surgeons toward a more durable treatment of combined aortic valve and ascending aortic disease.

For ten or more years before this report, the lesions had been handled by aneurysm plication and banding, graft replacement, and attempts to conserve the true aortic root and valve. The basic pathological process was yet to be clarified. There was much discussion over management and all authors seemed to report difficult early and late operative complications. In 1964, Groves and associates emphasized the important relationship of the aneurysm and the aortic valve and the need to replace the valve to prevent further dilatation of the aortic annulus. In a discussion at the time, Dr Groves hoped that in the future he would have the courage to anastomose the coronary ostia to the graft. In 1964 and again in 1971, Wheat and co-workers presented a more complete removal of aortic root tissue but still with separate aortic valve and graft suture lines. The complication rate remained high in these patients, many of whom had cystic medial necrosis of the aortic root. Bentall's procedure went the last step, a more complete replacement, and hopefully a more durable repair (Figs 2, 3). His report in 1972 on 10 patients had an operative mortality of 20%. In 1970 Edwards successfully used Bentall's technique on 3 patients, without mention of a prior work.

Most of the potential advantages of Bentall's procedure were mentioned in his initial brief report and they have remained unchanged. Such a worthy contribution in a foreign journal was not so readily accepted. There seemed an unreasonable reluctance to apply this technique even after others began to use it with surprising success. Many felt compelled to remove old efforts from their files to support the adequacy of separate management of the valve and aorta. Alas, the short-term and long-term complications of operations for residual aortic root problems made for fascinating reading. On occasion, while trying to present a case for separate aorta and valve management, such authors demonstrated the superiority of the more complete method of replacement, even in terms of early operative mortality.

If the procedure seemed as important as some thought, several problems needed to be overcome. One was continual refinement and learning of the surgical procedure. The other concerned when and in whom it should be

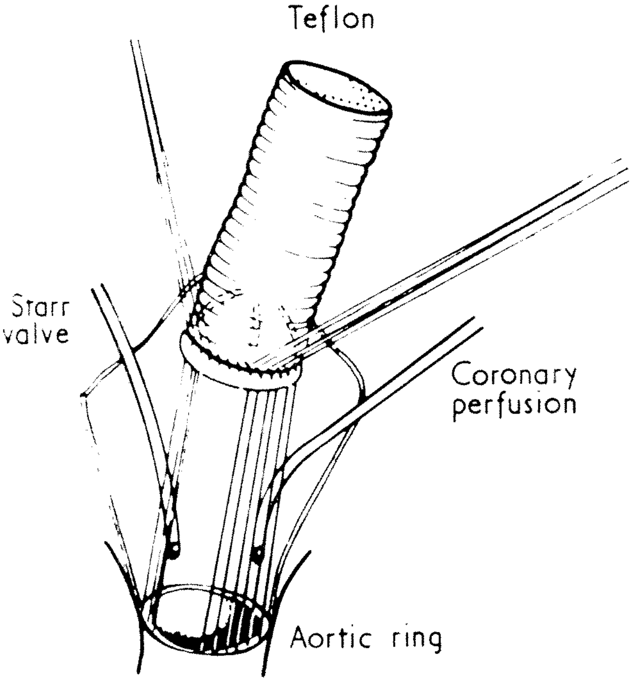


Fig 2. Starr valve has been sutured to aortic prosthesis: sutures have been placed in aortic ring before fixing the combined prostheses. (Reprinted from [5] by permission of the British Medical Association.)

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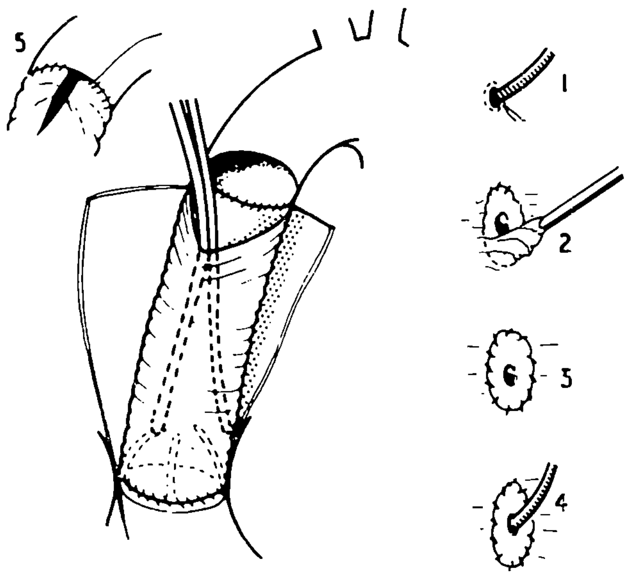

Fig 3. Combined prostheses in situ. Insets 1 to 4 show details of holes fashioned in the side wall of the Teflon tube to reincorporate the coronary ostia within the lumen of the new ascending aorta. Inset 5 shows the vertical slit in the prosthesis. (Reprinted from [5] by permission of the British Medical Association.)

applied. The patient with true Marfan's syndrome is the most direct beneficiary of this information, which, it was hoped, would prevent early death. Other patients with the same disease, but lesser forms, have also benefited. Improved grafts, sutures, cardioplegia, albumin baking, and instruction have made for continued improved surgical results and have benefited many patients. Although the potential market for a manufactured prosthesis was possibly not very great, the Shiley Company graciously helped create such a graft. The initial graft seemed bulky, as the sewing ring remained within the Dacron tube. Others used this prosthesis, but it was not until a tapered graft with a totally external sewing ring was quickly put together for a specific patient that a very good manufactured product appeared. To have a good prosthetic device available on the shelf has been a valuable addition, and these remain available from most valve manufacturers. It seemed proper to call this a composite graft.

It would appear that the long-term results will prove satisfactory. Many patients who have been operated on have maintained a stable aortic root replacement for 15 to 20 years now and continue to be followed for other potential complications of cystic medial necrosis. Applications of the procedure have become much more accepted in younger patients with Marfan's syndrome, in some acute dissections, and in cases of left ventricular–aortic discontinuity secondary to aortic root endocarditis and abscess. The homograft aorta has become available and appears to have a distinct place, but may not maintain the same durability. It has been enjoyable to see this short contribution in 1968 gradually gain acceptance by surgeons and even by medical colleagues as well. It appears that in 1990 when a composite graft is on the surgical schedule, everyone has a very good idea of what one is trying to accomplish.

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Mediastinal Epithelioid Hemangioendothelioma

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An epithelioid hemangioendothelioma arising from the innominate vein in the anterior superior mediastinum of a 62-year-old man was treated by wide local excision followed by radiotherapy. This clinicopathologic entity is a rare cause of mediastinal mass and is characterized by an intermediate malignant potential. To our knowledge, very few cases have been reported in the mediastinum; those cases are listed in this report. Gross and microscopic distinguishing features of epithelioid hemangioendothelioma, as well as differential diagnosis, are reviewed from the literature. Finally, clinical behavior is discussed and treatment options are suggested.

(Ann Thorac Surg 1990;49:680–5)

Thymic and neurogenic tumors are the most common primary neoplasms in the mediastinum. Mesenchymal tumors account for nearly 5% to 10% of mediastinal tumors [1]. Of these, tumors of blood vascular origin remain relatively infrequent, especially when compared with their incidence in other parts of the body. These tumors include hemangiomas, frequently of a cavernous nature, benign or malignant hemangiopericytomas, hamartomas, and hemangioendotheliomas [2]. This is a report of a case of mediastinal epithelioid hemangioendothelioma in a 62-year-old man. Few similar clinicopathological entities have been reported [3, 4]. A review of the literature on this subject is included.

Case Report

The patient was a 62-year-old man with a long history of chronic obstructive pulmonary disease, who was seen with increasing dyspnea on exertion and cough productive of purulent sputum. He denied any hemoptysis or recent weight loss. He had a smoking history of more than 80 pack-years. His medications on admission consisted of theophylline and metaproterenol inhaler. There was no family history of solid tumors. Physical examination revealed diffuse bilateral expiratory wheezes on lung auscultation. The rest of the examination, including head and neck, was within normal range. A hematologic profile, electrolyte studies, and liver function test results were unremarkable.

A chest radiograph was obtained revealing a homogeneous noncalcified 7-cm rounded mass in the anterior mediastinum (Fig 1). A radiograph taken 2 years earlier had no evidence of a mass. A computed tomographic scan of the chest showed the mass to extend toward the left upper lobe and the aortic arch. Fungal serology and PPD skin test results were negative. Preoperative bronchoscopy was normal. Two successive percutaneous fine needle biopsies demonstrated spindle-shaped and epithelial malignant cells, but no definitive diagnosis could be made. Preoperative impression was that of a malignant thymoma.

The patient underwent a median sternotomy in February 1984, with removal of a 10 × 9 × 7-cm grossly circumscribed anterior mediastinal mass arising from the innominate vein and extending down to midcardiac level. The tumor was removed in its entirety along with the innominate vein. No adenopathy was noted.

The tumor weighed 120 g and was grossly a firm brown ovoid solid mass. Hematoxylin and eosin, trichrome, reticulin, and periodic acid-Schiff stains showed a locally infiltrative tumor composed predominantly of epithelioid spindly cells arranged in whorls, strands, and nests, with acidophilic epithelial-appearing cytoplasm. The nuclei were vesicular and showed occasional intranuclear blebs of cytoplasm. Diffuse myxoid areas were noted with foci of metaplastic cartilage and bony spicules. Areas of geographic coagulative necrosis were noted. Only rare mitoses were encountered. There was a sprinkling of lymphocytes throughout the tumor (Fig 2). Ultrastructural electron microscopic studies showed a superabundance of intracytoplasmic fine filaments as well as distinctive Weibel-Pallade bodies (Fig 3), characteristic of endothelial cells.

The patient's postoperative course was uneventful. He was treated with irradiation, receiving 4,500 rads by linear accelerator followed by 1,000 rads boost to the tumor bed. He remains well, free of recurrent disease, 4.5 years after resection.

Literature Review and Comment

The term *hemangioendothelioma* is used to describe a heterogeneous group of tumors intermediate between benign hemangiomas and malignant angiosarcomas. Three distinct subtypes of hemangioendotheliomas have been described, including epithelioid hemangioendothelioma (EH), spindle-cell hemangioendothelioma, and the so-called Dabska tumor [5]. Epithelioid hemangioendothelioma bears some resemblance to another benign entity, referred to as epithelioid hemangioma (also known as angiolymphoid hyperplasia with eosinophilia or Kimura's disease) and which typically occurs as reddish papules in the skin or subcutaneous tissue, most commonly in the

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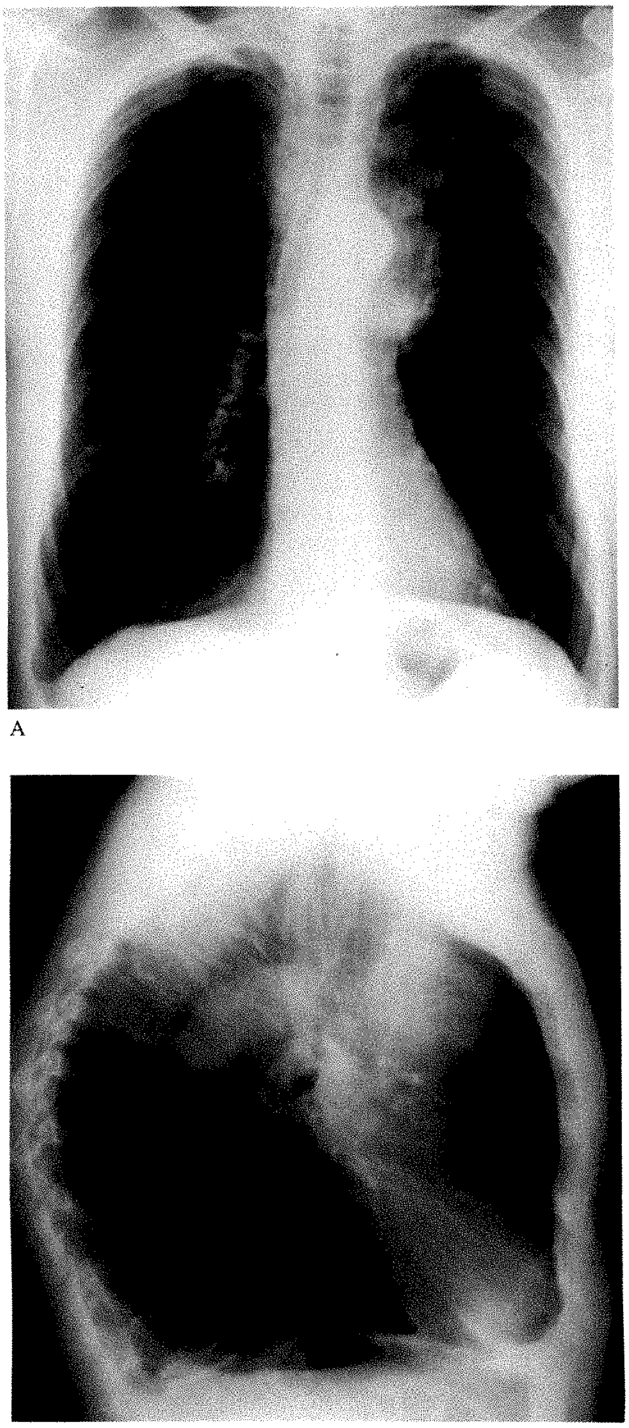

Fig 1. Posteroanterior (A) and lateral (B) chest radiographs showing a 7-cm spherical noncalcified soft-tissue mass in the anterior mediastinum. The anteroposterior diameter is increased and the diaphragm is flattened, consistent with chronic obstructive lung disease.

head and neck areas [6]. Some investigators have suggested that EH and Kimura's disease should be viewed within a unified spectrum, referred to as histiocytoid hemangiomas [7].

At the microscopic level, EH is characterized by a subtle degree of vascular differentiation, manifested mainly by the formation of small intracellular lumina as primitive vascular spaces. The tumor has spindly epithelioid cells arranged in nests or cords within a myxoid matrix rich in sulfated acid mucins. There are no well-formed vascular spaces. When such a pathological diagnosis is being considered, the endothelial nature of the tumor may be confirmed by a positive immunohistochemistry stain for Factor-VIII–related antigen [4, 5] or lectin binding for *Ulex europaeus* antigen. Electron microscopic studies often demonstrate the presence of intracellular Weibel-Pallade bodies, characteristic of endothelial-derived cells.

Epithelioid hemangioendothelioma has been reported in patients over a broad age range. It has also been described in a variety of superficial and deep body areas [8]. Location in soft tissues is particularly prevalent, especially in the extremities. There is no sex predilection in soft-tissue EH, which is typically seen as a painful tender mass that has been present for a variable period of time [4]. More than 50% of soft-tissue EHs reported to date arise from a blood vessel, commonly a medium-sized or large vein, such as the femoral, iliac, or jugular vein [4, 5]. In such situations, the tumor appears to expand the vessel without destroying its basic architecture. It is rarely grossly hemorrhagic, as opposed to hemangiomas and angiosarcomas. In the patient described in this report, the tumor arose from the innominate vein.

Epithelioid hemangioendothelioma has also been described in the lung parenchyma, where it was previously referred to as intravascular bronchioloalveolar tumor; in this location, it typically is seen in young women as numerous bilateral pulmonary nodules less than 2 cm in size, mimicking metastatic disease [9–11]. Furthermore, EH has been noted in the liver, where it shows again a predilection for women, and needs to be differentiated from sclerosing cholangiocarcinoma [12]. Epithelioid hemangioendothelioma also occurs in bone, where it typically tends to affect younger men, is often multicentric, and is also referred to as angioglomoid tumor [13]. The tumor also develops in the head and neck areas, especially in the submandibular region [14]; differentiation from Kimura's disease needs to be made in these cases. Also, EH can occur with numerous thoracic manifestations including mediastinal mass, diffuse pleural thickening, and lymphangitic spread [3].

To our knowledge, there have only been scattered reports of hemangioendothelioma seen as a mediastinal mass (Table 1). In their 1963 review of the literature to that date, Pachter and Pattes [2] believed that only 8 acceptable cases of mediastinal hemangioendotheliomas had been described. Of the cases listed in Table 1, only the first 3 are subspecified as epithelioid hemangioendothelioma; the rest are only labeled as hemangioendothelioma, with no specification of subtype. All cases reported as occurring in young children were located in the posterior mediastinum. Although the numbers are too small to allow any significant conclusions, there does not appear to be any sex predilection for mediastinal EH. The age range is very large. None of the reports makes mention of distant extramediastinal metastasis, although one did describe local extension from the posterior mediastinum into the

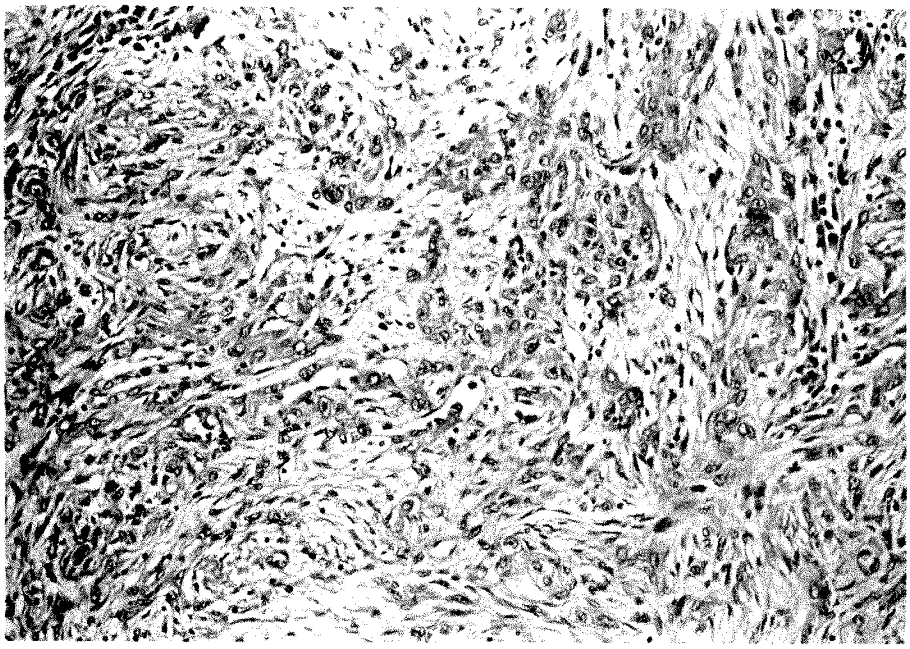

Fig 2. Characteristic histology of epithelioid hemangioendothelioma. High-power light micrograph of sections showing atypical epithelioid and spindly tumor cells with occasional formation of abortive vascular lumina. (Hematoxylin and eosin, ×520 before 28% reduction.)

deep soft tissues of the neck in an infant [15]. Follow-up time is too variable to allow any meaningful conclusions.

The symptomatology resulting from mediastinal hemangioendothelioma or EH cannot be expected to be different from that of other mediastinal masses in a similar location; dyspnea, stridor or wheezing from airway obstruction, cough, chest pain, dysphagia, and conceivably other symptoms such as vocal cord paralysis may be possible.

Hemangioendothelioma in the mediastinum can be confused on clinical grounds with a wide variety of more common mediastinal masses. In children, especially in the posterior mediastinum, neurogenic tumors are statistically the most likely [18]; congenital cysts and lymphomas are also quite common. In adults, in the anterior-superior mediastinum, thymomas, thyroid lesions, and lymphomas are more likely than tumors of vascular origin. In the middle mediastinum, pericardial and bronchogenic cysts have to be considered in the differential diagnosis. Finally, in the posterior mediastinum, neurogenic tumors and gastroenteric cysts should be kept in mind [20].

Radiologic studies, especially computed tomographic scanning, can sometimes be useful to tentatively exclude certain causes of mediastinal masses that have typical radiographic features, in addition to helping determine local extent of the lesion as well as plan the best surgical

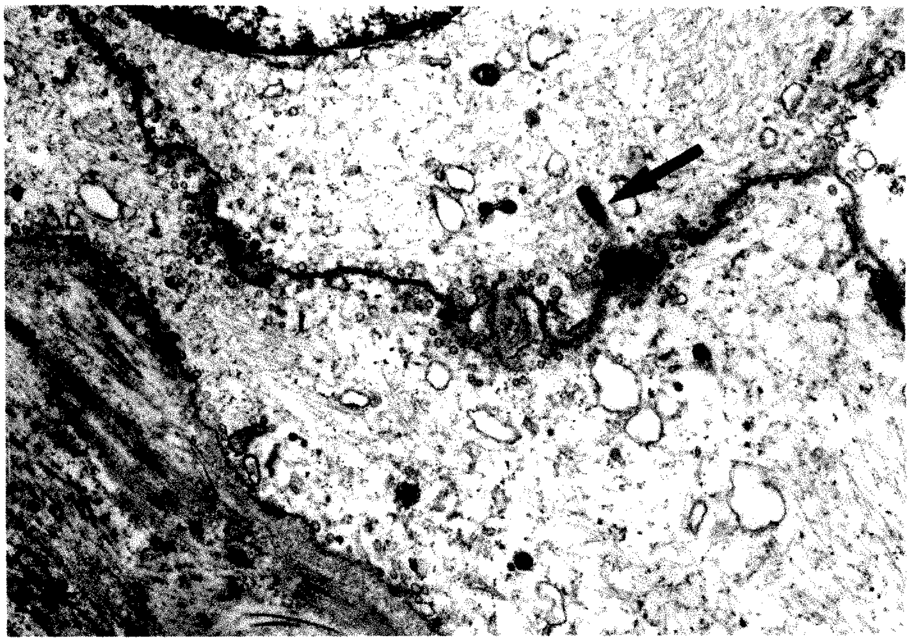

Fig 3. Electron micrograph showing cytoplasm of two tumor cells with apposing cell membranes (across center of field). Note the irregular membrane contour with numerous phagocytic vacuoles and junctional complexes. Tumor cell cytoplasm contains a membrane-bound organelle called a Weibel-Pallade body (arrow), *characteristic of endothelial differentiation. (Uranyl-acetate lead-citrate, ×18,000 before 28% reduction.)*

Table 1. Summary of Data on Patients with Mediastinal Epithelioid Hemangioendothelioma and Mediastinal Hemangioendothelioma

Patient No.	Reference	Year	Age	Sex	Location in Mediastium	Type	Treatment	Follow-Up
1	Toursarkissian et al (present report)	1990	62 yr	M	Anterior-superior	EH	Wide local excision with postoperative radiotherapy	NRD 4.5 yr after treatment
2	Yousem et al [3]	1987	35 yr	F	Anterior-superior	EH	Local excision	NRD at 2 mo follow-up
3	Weiss et al [4]	1982	NS	NS	NS	EH	NS	NS
4	Tarr et al [15]	1986	3 mo	F	Posterior	H	Combination chemotherapy; biopsy only	Tumor with slight shrinkage, 4 mo into treatment
5	Awotwi et al [16]	1983	Newborn	M	Posterior	H	Incomplete surgical resection	Tumor not extending at 3 yr follow-up
6	Awotwi et al [16]	1983	5 mo	NS	Posterior	H	Complete local excision	NRD at 6 mo follow-up
7	Bedros et al [17]	1980	5 mo	F	Posterior	H	Incomplete surgical resection	Tumor not extending at 14 mo follow-up
8	Heimburger et al [18]	1965	3 yr	F	Posterior	H	Operation with postoperative radiotherapy	NRD 4.5 yr after treatment
9	Pachter and Paltes [2]	1963	66 yr	M	Anterior	H	Incomplete surgical resection with postoperative radiotherapy	Died 7 mo postoperatively; no evidence of metastases
10	Pachter and Paltes [2]	1963	66 yr	M	Middle	H	Incomplete surgical resection with postoperative radiotherapy	Died 2 yr postoperatively; no evidence of metastases
11–18	Pachter and Paltes [2]	pre-1963	18–62 yr	M-F	7 anterior, 1 posterior	H	Various therapies, including operation, radiotherapy, and nitrogen mustard	Five died within 2–16 months following treatment; 1 with no follow-up; 1 alive 3 yr postoperatively; 1 not noted in report
19[a]	Balbaa et al [19]		18 yr	M	Posterior	H	Operation with postoperative radiotherapy	NS

[a] Not noted as hemangioendothelioma in Pachter and Paltes' 1963 review of the literature on mediastinal hemangioendothelioma [2].

EH = epithelioid hemangioendothelioma; H = hemangioendothelioma; NRD = no recurrent disease; NS = not specified.

approach. For instance, in 1 case of hemangioendothelioma in the posterior mediastinum of a 3-month-old infant [15], the finding on computed tomographic scan of smooth well-defined margins and homogeneous enhancement was considered an argument against the possibility of primary thoracic neuroblastoma. However, there have been no reports commenting on radiographic findings that can be considered pathognomonic for hemangioendotheliomas.

The intraoperative finding of a tumor originating from a vessel should raise the possibility of EH as one of the various tumors of blood vascular origin. In addition to EH and hemangioendotheliomas, these include hemangiomas of various kinds, hemangiopericytomas, and angiosarcomas. Leiomyomas and other sarcomas can also occasionally arise from the walls of large blood vessels; primary sarcoma of the aorta is an example of such. Hemangiomas are usually more common in superficial tissues, but there have been reports of this tumor occurring in the mediastinum [21]. Angiosarcoma also occurs in skin and soft tissues. Up to 3% [22] develop from great vessels, and it has been reported to arise from the inferior vena cava and aorta, as well as the superior vena cava [23]; these tumors are frequently highly hemorrhagic as well as deeply invasive. Mediastinal angiosarcomas are very malignant tumors that need to be differentiated from epithelioid hemangioendothelioma. Hemangiopericytoma is also a rare mediastinal tumor; it is frequently encapsulated and of an often solid, gray to white quality [24]. Usually, EH that arises from a vessel has a variegated white to red coloration.

Histologically, the tumor can be confused with metastatic adenocarcinoma [8], with the primitive intracytoplasmic channels being confused with mucin vacuoles. Careful staining techniques, immunohistochemical studies for Factor-VIII–related antigen, and electron microscopic studies should, however, leave no doubt as to the diagnosis. Myxoid chondrosarcoma as well as various sarcomas that assume an epithelioid appearance are lesions that should also be microscopically distinguished from EH.

The clinical behavior of EH has been described as intermediate between that of the benign hemangioma and the malignant angiosarcoma [4, 5]. In their review of soft-tissue EH, Enzinger and Weiss [5] noted an overall local recurrence rate of 13% within 48 months, a metastasis rate of approximately 31%, and a mortality rate of 13%. In addition to the gross appearance of deep invasion and other features that can be noted intraoperatively, certain histological findings have been reported as useful in predicting malignant behavior [4, 5, 8]. These include, most importantly, a number of mitoses exceeding one to two per 10 high-power fields, the presence of necrosis, metaplastic bone, and increased cellular pleomorphism. Tumors that exhibit these characteristics are more likely to metastasize and cause the eventual death of the patients. For instance, the rates of metastases and death from tumor for histologically "malignant" forms have been reported as 53% and 31%, respectively, as compared with 17% and 3% for the histologically "benign" varieties [8]. It is important to note that these features are not absolute, and that many histologically "benign" tumors may behave in a clinically malignant fashion. Equally noteworthy is the fact that less than half of all patients in whom metastases develop actually die of the disease, as many of these metastases can be cured [8]. The prognosis is much less favorable in cases occurring in the lungs or liver, where the mortality can be as high as 35% and 65%, respectively [5]. In intravascular bronchioalveolar tumor, the presence of clinical symptoms, local spread, and regional lymphadenopathy have been noted to be poor prognostic signs [9]. The scarcity of reports on mediastinal EH does not allow us to comment on recurrence rates or metastasis rates, although we have no reason to suspect that the behavior of EH in the mediastinum should be any different from its behavior in other soft-tissue parts of the body. The patient described by Yousem and associates [3] was free of recurrent disease at 2 months of follow-up, and the patient in the present case report is also free of recurrence 4.5 years after treatment.

In soft-tissue EH, Enzinger and Weiss [5] have recommended that benign-appearing EH be treated by wide local excision without adjuvant therapy, and that more histologically malignant varieties be treated in a manner similar to high-grade angiosarcomas with radical local excision and possibly adjuvant chemotherapy. In the case of mediastinal EH reported by Yousem and associates [3], the tumor was circumscribed and contained some stromal calcification; the patient was treated by local resection. In the case described in the present report, the histological material demonstrated necrosis as well as metaplastic cartilage and bone, two features noted as possible indicators of malignancy; in view of this fact, it was elected to treat the patient postoperatively with irradiation after the local excision. We believe that intraoperative frozen-section studies by the surgical pathologist are helpful in determining the extent of local resection needed and ensuring that margins of resection are free of tumor. Adequate consideration should be given to the resection of regional lymph nodes for the evaluation of possible metastatic disease. To our knowledge, there have been no studies documenting chemotherapeutic sensitivities of EH; agents used for the treatment of angiosarcomas may be considered. There is a report of a case of unresectable posterior mediastinal hemangioendothelioma in a 3-month-old girl that was treated with a combination of prednisone, actinomycin D, and vincristine, with slight shrinkage of the tumor at 4 months of follow-up [15].

In summary, EH is an infrequently noted cause of mediastinal mass with intermediate malignant potential. We report 1 case arising from the innominate vein in the anterior-superior mediastinum. Vascular origin of a mediastinal mass should raise the consideration of EH as one of the possibilities in the differential diagnosis. Wide local excision has been used for histologically benign forms. For more malignant-appearing tumors, treatment plans must be individualized.

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CORRESPONDENCE

Traumatic Rupture of the Thoracic Aorta

To the Editor:

I read with interest the two articles in the July 1989 issue of *The Annals* concerning traumatic rupture of the thoracic aorta. The first is by Hess and associates [1] and represents another beautiful contribution that shows, in a small series of patients, that better results can be obtained when distal perfusion is provided.

The second is an editorial by Mattox [2]. Dr Mattox is to be congratulated for emphasizing the extreme difficulty and the potential danger of treating traumatic rupture of the aorta for the "casual thoracic surgeon." I certainly agree with most of his arguments; however, I cannot share his fatalism about the so-called "unavoidable" 5% to 7% incidence of paraplegia.

My experience in descending thoracic aortic aneurysms comprises 226 surgically treated cases. The incidence of paraplegia is 0%. Thirty patients were treated for an acute aortic transection. All patients were protected with a single method of distal perfusion, a 9-mm Gott shunt used as an external bypass between the ascending and the descending aorta [3].

The proper position and the performance of the shunt were constantly monitored with a flow meter. Shunt flows varied from 1,800 to 4,900 mL/min, and averaged 3,050 mL/min. Blood reinfusion, when needed, was assured with an autotransfusion unit operated by a pump technician. The aortic cross-clamping ranged from 18 to 73 minutes and averaged 37 minutes for the 30 patients; it was reduced to 27 minutes for the last 10 patients. A survival rate of 93% (28/30) was obtained. No renal failure, no paraplegia, and no other spinal cord ischemic deficit occurred. I hope this experience meets Dr Mattox's criteria for surgical expertise and allows me a few practical nonmedical-legal thoughts.

Aortic transection involves only a very limited segment of the descending aorta that usually does not include the critical sources of spinal cord blood supply. Therefore the most important factor responsible for paraplegia is the lack of adequate distal aortic perfusion. For the past 75 years, substantial and contributive experimental and clinical works have clearly established that the tolerable ischemic time for the spinal cord is around 15 to 18 minutes [4, 5], that the occurrence of paraplegia increases with longer aortic cross-clamping times reaching incidences up to 25% in experienced hands [6], and that the use of an efficient technique of distal perfusion decreases its incidence [7–14].

I agree that the use of a shunt complicates the surgical procedure. However, its use without systemic heparinization offers three major advantages: (1) a remarkable cardiovascular and metabolic stability, (2) preservation of coagulation factors, and (3) conservation of spinal cord perfusion.

This physiological approach based on the principle of organ protection should allow more patients to survive the surgical repair with an intact cord. If an inadvertent paraplegia nevertheless occurs, then it could be ultimately tagged "accidental," due to an unusual anatomical distribution of spinal cord blood supply.

Paraplegia being such a disastrous complication, it is my opinion that a randomized series is not necessary because it would inexorably redemonstrate what has been already documented in the past. I agree with Mattox that the surgical treatment of an aortic tear is most difficult and should only be undertaken by an experienced surgeon with optimal staffing and technical and laboratory support available around the clock. Most patients can tolerate the unavoidable delay to reach optimal resources.

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Reply

To the Editor:

Dr Verdant has achieved remarkable results using the Gott shunt to manage patients with transected aortas. Regardless of the technique used, heparinless bypass provides protection to distal tissues, aids spinal cord perfusion, and simplifies the conduct of the operation. The Gott shunt, in our hands, seemed more cumbersome to use and we found it easier to control shunt flow

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and proximal pressures with the Bio-Medicus pump and left atrial–femoral bypass. With experience, either technique provides excellent results. I thank Dr Verdant for his comments regarding our paper.

My colleagues and I echo Dr Verdant's comments regarding the editorial by Dr Mattox. Our current operative strategy evolved from observations of patient response to different techniques rather than concern for medical-legal implications. We do agree with Dr Mattox that seriously injured patients should be transported primarily to major trauma centers. In our small series, 85% of patients with this injury admitted directly to our trauma center survived versus 54% transferred from other hospitals.

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Reply

To the Editor:

I fully recognize Dr Verdant's excellent results using a temporary bypass shunt in lesions of the descending thoracic aorta. Among his 226 thoracic aortic aneurysms, just over 10% were acute traumatic lesions. Dr Pate of Nashville, using cardiopulmonary bypass, and we in Houston, using clamp repair, have results identical to those of Dr Verdant, ie, 0% paraplegia rate. My citation of a 5% to 7% paraplegia rate is derived from compilation and analysis of data in the literature, including a recent large series presented at the American Association for Thoracic Surgery, with a greater than 20% paraplegia rate.

Dr Verdant correctly points out that use of the shunt requires more expertise than the clamp/repair technique, and that, if possible, patients with aortic injury should be treated at regional trauma care facilities. Unfortunately, this is not always possible.

The primary purpose of my editorial was to address the medical-legal aspects of aortic trauma which, unfortunately, threaten to jeopardize the patient's access to surgical care at the time when it is most critical.

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Retrograde Coronary Sinus Cardioplegia in Redo Aortocoronary Bypass

To the Editor:

In the October 1988 issue of *The Annals*, Snyder and associates [1] advocated the use of retrograde coronary sinus cardioplegia during redo coronary artery bypass grafting. They reported excellent results with this method of myocardial protection in redo cases, but were unable to confirm washout of atheromatous debris with the technique. Certainly atheroembolism [2] resulting in the "trash heart" [3] is a major concern in redo coronary bypass procedures. Despite extreme care in dissection, early ligation of previous grafts, and delay in dissecting the heart until bypass, distal embolism may still occur.

We recently encountered 2 cases that demonstrate this complication and also correct a common misconception that totally occluded grafts do not cause distal embolism. Both cases involved patients with previous multiple bypass grafts (three and four) that were all totally occluded, moderately impaired ventricular function, and accelerated angina. We used retrograde coronary sinus cardioplegia to improve myocardial protection. Our method includes (1) bicaval cannulation with snares, (2) injection of a single large bolus (700 to 1,000 mL) of crystalloid aortic root antegrade cardioplegia immediately after aortic cross-clamping, (3) direct cannulation of the coronary sinus with a balloon-tipped catheter (DLP Corporation) via a small atriotomy secured by a tourniquetted pursestring suture, and (4) continuous infusion of crystalloid or blood cardioplegia with aortic root venting. The previously described precautions were taken, including proximal ligation of all grafts during aortic dissection or immediately after the initial antegrade cardioplegic injection. In both patients, several atheroembolic particles were washed out retrogradely after two separate, previously grafted, arteries immediately distal to the old graft insertion sites were opened. Both patients received four new bypass grafts and recovered uneventfully, with no evidence of perioperative myocardial infarction.

Since 1986, we have increasingly used retrograde coronary sinus perfusion with both blood and crystalloid cardioplegia in complex valvular, high-risk coronary artery bypass, and redo coronary artery bypass operations with gratifying results. We are now of the opinion that retrograde coronary sinus cardioplegia should be strongly considered for all redo coronary bypass operations. The method affords excellent myocardial protection, is only minimally cumbersome, and can wash out distal embolic debris that may occur despite all precautions.

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Reply

To the Editor:

Since our report was published, my colleagues and I have continued to use this method of cardioplegia delivery in redo coronary operations, especially in the presence of diseased saphenous vein grafts. We have observed washout of atheromatous material from the distal coronary bed in several cases. It is our practice, however, to avoid using antegrade cardioplegia in situations in which atheromatous disease of vein grafts is present.

We have not observed any atheromatous material washing out from coronary arteries where the vein grafts were totally occluded and ordinarily do not use retrograde cardioplegia delivery when all the veins are obstructed.

We appreciate very much the comments of Dr Calhoun and his associates and agree with them that retrograde coronary sinus cardioplegia should be strongly considered for all redo coronary bypass operations in patients with patent diseased veins.

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Thymectomy for Myasthenia Gravis

To the Editor:

Mulder and associates [1] describe the results of thymectomy for myasthenia gravis employing a transsternal procedure. In their recent series of 84 patients, "30 (36%) went into remission during a total of 3,015 patient-months of follow-up." The remission rate, calculated on the basis of rate per 1,000 patient-months, was 9.95. Referring to the transcervical-transsternal "maximal" thymectomy that we have described [2, 3], they state "it is considered imprudent to radically explore and excise contiguous tissues high in the mediastinum and in the neck, as the yield of aberrant thymic tissue is low, and in our opinion, it has not been established that our results are inferior to those of the advocates of the more radical procedure."

The results after transcervical-transsternal "maximal" thymectomy refute the above statements. First, our anatomical studies [2] demonstrated thymic tissue outside the two cervical lobes in 32% of the cases and in the region of the aortopulmonary window in 24% of the cases. Second, using the remission rate per 1,000 patient-months calculations as described by Mulder and associates, in our series of 72 patients [3] who had had a "maximum" thymectomy for myasthenia gravis, 33 (46%) went into remission during a total of 2,374 patient-months of follow-up. The rate of remission calculated on the basis of number per 1,000 patient months was 13.9.

Accordingly, it can be seen from this analysis, as well as other analyses previously reported [3], that the procedure we have described removes more thymic tissue and gives better results than lesser resections, including those of Mulder and associates [1]. Our findings support the thesis that the more complete the thymectomy, the better the results.

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Reply

To the Editor:

My colleagues and I appreciate the comments by Dr Jaretzki concerning our recent report. His detailed histological analyses of the thymic anatomy including that of aberrant tissue found in contiguous or more remote areas in the cervical or mediastinal regions are important informational observations. Our current knowledge about myasthenia gravis, incomplete as it may be, supports the logic of removing as much thymic tissue as possible. However, we agree with Dr Jaretzki's previous publication that no matter how extensive the surgical procedure may be, some residual microscopic foci or distant ectopic tissue may remain even after "maximal" thymectomy. Therefore the real issue is whether the more aggressive approach of a transcervical-transsternal "maximal" thymectomy justifies the additional patient risk and morbidity over that of the transsternal procedure we have described.

Obviously Dr Jaretzki is convinced that the results following his procedure are superior to those we have reported. In the paper from which he refers to a 46% remission rate in 72 patients, it is disconcerting to note that there were 124 patients who had a "maximal" thymectomy of whom only 95 were evaluated. It is not clear how this selection was made. In addition the remission rate of 46% was calculated on 72 patients without thymoma, but the remaining 23 patients (8 reoperations and 15 with thymomas) were not included in the calculations. Thus he compares our remission rate of 36% for our entire series of 84 patients to his 46% remission rate in one group of 72 patients out of a total series of 95 patients. If one calculates the remission rate for his total series it is 37.8%, which is not statistically different from ours. Data are not available in his article to calculate the remission rate per 1,000 patient-months follow-up for the 95 patients, which would then need to be compared with our rate of 9.95.

At this point we remain convinced that the procedure we have described achieves long-term results comparable with those seen after the "maximal thymectomy," and with less morbidity and potential risk to the patient.

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Southern Thoracic Surgical Association Rules for Submission of Abstracts

Abstracts for papers to be presented for the 1990 Annual Meeting are now being accepted. The deadline for postmark of abstracts is May 15, 1990. An original and 1 copy should be submitted, using the official Association abstract form, to Donald C. Watson, Jr, MD, Chairman, Program Committee, The Southern Thoracic Surgical Association, 111 E Wacker Dr, Chicago, IL 60601. Additional forms are available upon request from the Association.

Abstracts must summarize an original contribution not presented or accepted for presentation elsewhere. *Abstracts received after the deadline will not be considered.* The information requested at the bottom of this form must be completed indicating who is to receive the notice of acceptance or rejection and who will present the paper if accepted.

Abstracts may be submitted for the regular program, the Forum, or either. Papers selected for the Forum will be published in *The Annals of Thoracic Surgery* if they pass the usual editorial review.

Essayists are reminded that all papers accepted for presentation must be submitted in triplicate to Gordon F. Murray, MD, Secretary-Treasurer, at the time of presentation or to the Editor of *The Annals of Thoracic Surgery* prior to presentation.

Abstracts frequently obscure the value of their content because they are poorly organized. It is important that methods and results be clearly defined in the abstract; general statements such as "results will be presented" are not acceptable. When a procedure or method is stated to have "advantages," those advantages should be defined. Abstracts that simply summarize results without defining objectives and methods are equally undesirable.

Presentations in the regular program are limited to 10 minutes; those in the Forum are limited to 5 minutes.

In discussion of papers, the Association encourages a healthy spirit of constructive critical review and rebuttal by comments pertinent to the content of the paper being presented. Presentation of the discussion in the form of "secondary papers" will not be welcomed.

INSTRUCTIONS FOR ABSTRACTS

The official abstract form or photocopies must be used. **Authors must indicate whether they want the abstract considered for the regular program, the Forum, or either by checking the appropriate box. Authors must also categorize the principal subject of their abstract by checking the appropriate box.**

1. Title of abstract must be completely CAPITALIZED.
2. Full names of authors must appear on the abstract, without degrees, titles, or appointments.
3. Indicate each author who is a member of the Southern Thoracic Surgical Association with an asterisk following the name.
4. Underline the name of each STSA new member (elected to membership in 1989).
5. Institution or address is to be given.
6. Abstracts must be limited to 200 words and *must be typed double-spaced.*
7. When percentages are used, the absolute numbers of derivation must be stated.
8. The body of the abstract should include:
   - a. A brief statement of the purpose of the study, referring, as appropriate, to the current state of research in the field.
   - b. The methods of study or experimental approach clearly defined.
   - c. A summary of the results and/or conclusions of the study, including sufficient details to support those conclusions.
   - d. A statement concerning the significance and implications of the work.
9. The form must be signed by the senior author attesting that the material will not be presented elsewhere if accepted for the scientific program nor published elsewhere if accepted for publication in *The Annals of Thoracic Surgery.*

THE FOLLOWING INFORMATION MUST BE COMPLETED:

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THE
ANNALS OF
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Official Journal of The Society of Thoracic Surgeons
and the Southern Thoracic Surgical Association

EVENTS OF INTEREST

Brief notices of meetings, courses, and symposia relevant to cardiothoracic surgery are published as a service. They should be formatted as follows: meeting title, date, place, sponsor, accreditation, director, fee, minimum/maximum attendance, and address/phone for further information. Announcements should be sent at least 4 months in advance of publication to *The Annals of Thoracic Surgery*, 3108 Queeny Tower, Barnes Hospital Plaza, St. Louis, MO 63110-1041; telephone, (314) 361-6084.

Postgraduate Course in General Thoracic Surgery, Boston, Massachusetts—April 30–May 1, 1990

This course is sponsored by the Massachusetts General Hospital Thoracic Surgical Unit, under the auspices of the Harvard Medical School Department of Continuing Education, and is accredited for 16 hours in category 1 of the Physician's Recognition Award of the AMA. For information on this meeting, contact Harvard MED-CME, PO Box 825, Boston, MA 02117; or telephone (617) 432-1525.

Aortic Surgery Symposium II, New York, New York—May 4–5, 1990

This symposium is sponsored by Meadox Medicals, Inc, and is accredited for 15 hours in category 1 of the Physician's Recognition Award of the AMA and toward the New York Medical Association Certificate in Continuing Medical Education. For information, contact Beverly Farrier, Symposium Coordinator, ProMedica International, 620 Newport Center Drive, Suite 575, Newport Beach, CA 92660; or telephone (714) 640-5870.

Seventieth Annual Meeting of The American Association for Thoracic Surgery, Toronto, Ontario, Canada—May 7–9, 1990

For information on this meeting, contact American Association for Thoracic Surgery, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

Ninth Annual Educational Meeting of the Association of Physician Assistants in Cardiovascular Surgery, Toronto, Ontario, Canada—May 7–9, 1990

Held concurrently with the American Association for Thoracic Surgery Scientific Session, this meeting is accredited for 7 hours in category 1 of the Physician's Recognition Award of the AMA. For information on this meeting, contact the APACVS, 217 Hillcrest St, Box 74, Orlando, FL 32802; or telephone (407) 839-1752 (facsimile: (407) 422-0166).

Pulsed Lasers in Angioplasty, Tübingen, Federal Republic of Germany—May 11–12, 1990

This workshop is sponsored by the European Society of Cardiology, the Working Group of Coronary Flow and Angina Pectoris, and the European Association of Radiology. For information on this meeting, contact K. R. Karsch, Department of Cardiology, Medical Clinic, University of Tübingen, Otfried-Müller-Str 10, D-7400 Tübingen, Federal Republic of Germany.

Third International Symposium on Echocardiography and Doppler in Cardiac Surgery, Vienna, Austria—May 9–12, 1990

For information on this meeting, contact G. Maurer, MD, or W. Mohl, MD, PhD, c/o Interconvention, A-1450 Vienna, Austria; or telephone +43/1/23 69/2642 (telex: 11 18 03; facsimile: +43/1/23 69/648).

Eleventh Annual Scientific Session of the North American Society of Pacing and Electrophysiology, San Diego, California—May 31–June 2, 1990

For information on this meeting, contact the North American Society of Pacing and Electrophysiology, 13 Eaton Court, Wellesley Hills, MA 02181; or telephone (617) 237-1866 (facsimile: (617) 431-1991).

Eighth Annual Uniformed Services University of Health Sciences Cardiothoracic Surgery Symposium, Washington, DC—May 31–June 2, 1990

For information on this meeting, contact Fred H. Edwards, MD, Department of Thoracic Surgery, Walter Reed Army Medical Center, Washington, DC 20307-5001; or telephone (202) 576-1433.

The Society for Vascular Surgery/International Society for Cardiovascular Surgery, Los Angeles, California—June 4–6, 1990

For information on this meeting, contact the Association offices, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330.

Seventh International Congress Cardiostim 90, Nice, France—June 20–23, 1990

Cardiostim gathers several scientific events: Cardiostimulation 90 (cardiac pacing, electrophysiology, defibrillation), International Conference on High-Resolution ECG and Late Potentials, Fifth World Congress on Catheter Ablation, Cardio-Pharmaco 90, and Cardio-Rythmo 90. For information on this meeting, contact Secretariat Cardiostim 90, Department of Cardiac Pacing, Centre Chirurgical Val D'Or, 16, rue Pasteur, 92211 St. Cloud Cedex, France; or telephone (1) 46.02.70.72 (facsimile: (1) 46.02.05.09).

Sixteenth Annual Meeting of The Western Thoracic Surgical Association, Coronado, California—June 20–24, 1990

For information on this meeting, contact The Western Thoracic Surgical Association, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330.

Internal Thoracic Artery for Myocardial Revascularization, Toulouse, France—June 27–29, 1990

For information on this meeting, contact the Secretary of the Symposium, Department of Cardiovascular Surgery, Rangueil University Hospital, 31054 Toulouse Cedex, France; or telephone 61.32.26.51 or 61.32.26.53 (telex: 530 826 F).

Cardiac Arrhythmia Surgery International Seminar I, Geneva, Switzerland—June 27–29, 1990

For information on this meeting, contact Beverly Farrier, ProMedica International, 620 Newport Center Drive, Suite 575, Newport Beach, CA 92660; or telephone (714) 640-5870 (facsimile: (714) 759-6911).

Seventh Congress of the European Society of Biomechanics, Aarhus, Denmark—July 8–11, 1990

For information on this meeting, contact Seventh Congress of ESB, Biomechanics Laboratory, Orthopaedic Hospital, Randersvej 1, 8200 Aarhus N, Denmark; or telephone 45-616-7500, extension 4649.

Myocardial Preservation Into the 21st Century, Oxford, England—August 12–15, 1990

For information on this meeting, contact Beverly Farrier, ProMedica International, 620 Newport Center Drive, Suite 575, Newport Beach, CA 92660; or telephone (714) 640-5870 (facsimile: (714) 759-6911).

Thirty-ninth Congress of the European Society for Cardiovascular Surgery, Budapest, Hungary—September 9–12, 1990

For information on this meeting, contact Budapest Convention Centre, H-1444, PO Box 233, Budapest XI, Jagello u 1-3, Hungary; or telephone (361) 852-507 (facsimile: (361) 665-636; telex: 22-7717).

Fourth Annual Meeting of the European Association for Cardio-thoracic Surgery, Naples, Italy—September 17–19, 1990

For information on this meeting, contact the Organizing Secretariat, MCM, Rione Sirignano, 5, 80121 Naples, Italy; or telephone (0)81/7611085-7611277 (facsimile: (0)81/664372; telex: 710626).

Second Annual Meeting of the International Society for Applied Cardiovascular Biology, Venice Lido, Italy—September 17–20, 1990

For information on this meeting, contact the Congress Secretariat, Kele Teo, San Marco 4930, Venice, Italy.

Interim Meeting of The Society of Thoracic Surgeons, Chicago, Illinois—September 22–23, 1990

For information on this meeting, contact Richard P. Anderson, MD, The Society of Thoracic Surgeons, 111 East Wacker Dr, Chicago, IL 60601-4301; or telephone (312) 644-6610.

Mechanical Circulatory Support 1990, London, England—September 26–28, 1990

For information on this meeting, contact Anne Sumner, Mechanical Circulatory Support 1990, Medical Research Services, 60 Oxford St, Southampton SOl 1DL, Hants, England; or telephone 0703 231068.

Controversies in Cardiovascular Disease: Surgical Versus Medical Treatment, Aalst, Belgium—September 28–29, 1990

For information on this meeting, contact Dr F. Wellens, Department of Cardiovascular Surgery, O.L. Vrouwziekenhuis, Moorselbaan 164, B-9300 Aalst, Belgium; or telephone 053/78 53 53 (facsimile: 053/78 38 76).

CLASSIFIED ADS

Classified ads for positions wanted or available must relate only to the thoracic or cardiovascular surgical specialties. Display space (minimum, ¼ page) is also available through Media for Medicine, Inc, 440 Park Avenue South, New York, NY 10016 (telephone: (212) 684-5540; facsimile: (212) 545-0651). All ads are subject to the approval of the editor, and material deemed inappropriate for publication in *The Annals* will be rejected. If anonymity is desired, please indicate this at the time of submission and a code number will be assigned. All responses received will remain confidential in the publisher's office and will be conveyed to the advertiser shortly after receipt. The charge for all classified advertising is **$1.10 per word per insertion, minimum 20 words.** Abbreviations, dates, initials, post office box numbers, telephone numbers, years, and zip codes are considered one word each. **There is an additional fee of $15.00 per insertion for box number ads.** The copy deadline is 7 weeks prior to publication, eg, for the March issue, copy should be received by the 1st of January. Ad orders should be for a maximum of 6 months; ads may be renewed for up to 6 months at a time. Ad copy should be typed double-spaced and mailed in duplicate to: *The Annals of Thoracic Surgery*, Desk Editorial, Classified Ads, Elsevier Science Publishing Co, Inc, 655 Avenue of the Americas, New York, NY 10010 (facsimile: (212) 633-3695). Make *nonrefundable* check payable to Elsevier Science Publishing Co, Inc.

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SITUATIONS AVAILABLE

Cardiovascular and thoracic surgeon: Excellent opportunity for a board certified/eligible cardiovascular and thoracic surgeon to join a large cardiac surgery group in southern California. Must have a minimum of 1 year experience in pediatric and neonate cardiac surgery (including fellowship). Excellent fringe benefits. Respond with curriculum vitae.

Please respond to Box 144. 144E/D

Cardiothoracic surgeon wanted for a busy, expanding, 3-man, private practice group in the Midwest. Applicant must be ABTS eligible or certified. Prefer recent graduate with some background in electrophysiologic surgery. Please send curriculum vitae.

Please respond to Box 189. 189H/G

Cardiac-thoracic surgeon, needed for expanding program on west coast of Florida. Excellent opportunity, private practice, primarily adult cardiac including cardiac transplantation. BC/BE required. Please send CV.

Please respond to Box 226. 226K/D

Cardiothoracic surgeon, BE/BC, wanted to join surgeon in active cardiac/thoracic/vascular surgical practice in northern Alabama community of 80,000 (300,000 population draw). Clinical practice in two community hospitals. Lake/mountain recreation facilities. Generous compensation, future partnership.

Please write Sara Fleming, Business Manager, 801 Gaines Ave, Suite 305, Gadsden, AL 35903; (205) 492-9360. 237L/E

Associate Director for Heart Transplantation is urgently needed for rapidly expanding heart transplantation program in California.

Please respond with curriculum vitae to the Pacific Cardiothoracic Surgery Group, 201 S Alvarado St, Suite 626, Los Angeles, CA 90057. 240L/K

Cardiovascular and thoracic surgeon, board eligible/certified, wanted to join well-established private practice located in Ft. Lauderdale, FL. Send CV.

Please respond to Box 251. 251L/E

Cardiovascular and thoracic surgeon, board eligible or certified, wanted to join another surgeon in private practice in the Southeast. Please respond with curriculum vitae.

Please respond to Box 252. 252L/F

Thoracic-vascular surgeon wanted to join busy practice in Northeast. Board certified or eligible. Interested in practice of vascular and thoracic surgery. Excellent benefits package. Send curriculum vitae.

Please respond to Box 254. 254L/F

Cardiothoracic and vascular surgeon wanted to be first assistant. Twenty years experience preferred. Interested in practice of cardiac, vascular, and thoracic surgery. Excellent benefits package. Send curriculum vitae.

Please respond to Box 255. 255L/F

Cardiothoracic surgeon—to join a 2-man private practice in cardiac, thoracic, and vascular surgery in the Great Lakes region. Must be board certified or eligible. Recent graduates considered. Send curriculum vitae.

Please respond to Box 260. 260L/E

Cardiovascular/thoracic surgeon to complete 3-man group in adult cardiothoracic private practice in Michigan. Must be board eligible or certified. References required.

Please respond to Box 269. 269A/F

Cardiothoracic surgeon, board certified with 2 to 4 years experience in cardiac surgery, wanted to join a growing group of cardiac surgeons, cardiologists, and pulmonologists in the Pacific Northwest. General thoracic and vascular experience necessary. Research opportunities available, academic links possible. Salary leading to full partnership in 2 or 3 years; potential exists for leadership in major cardiac program.

Please send curriculum vitae to John R. Bauman, Administrator at The Thoracic Clinic, PC, 507 NE 47th Ave, Portland OR 97213-2282. 274A/F

Cardiovascular and thoracic surgeon: Position available for a board certified or board eligible to join practice in Northeast involved in adult cardiac, thoracic, and vascular surgery. Association will lead to partnership. Excellent fringe benefits. Please send curriculum vitae.

Please respond to Box 275. 275A/F

Thoracic surgeon: Excellent opportunity for experienced thoracic surgeon to join well-established hospital-based practice in general thoracic and vascular surgery. Hospital located in attractive New England coastal community and is affiliated with prestigious teaching institutions and medical schools.

Please contact Jeanne Dowrick, Fulton, Longshore & Assocs, 527 Plymouth Rd, Plymouth Meeting, PA 19462; telephone (800) 346-8397 or (215) 834-6780. 284B/D

Cardiovascular and thoracic surgeon, BC/BE, recently trained to join an established 3-man private practice group in Southeastern coastal capital city and two-university town. Great hunting, fishing, and sports. Submit CV and references.

Please respond to Box 293. 293B/E

A busy cardiothoracic group in the NYC area is looking primarily for a thoracic surgeon with potential for cardiac surgery in the future. Excellent salary and benefits. Respond with curriculum vitae.

Please respond to Box 211. 211J/D

Cardiovascular and thoracic surgeon: Position available for board certified or board eligible person to join busy practice involved in adult cardiac, thoracic, and vascular surgery in major New England city. Association will lead to partnership. Excellent fringe benefits. Please send curriculum vitae.

Please respond to Box 291. 291B/G

Cardiothoracic/vascular surgeon, board eligible/certified, wanted to join established group practice in Southeast. Please send curriculum vitae.

Please respond to Box 295. 295C/E

Cardiovascular and thoracic surgeon, BE/BC, to join another physician in a rapidly expanding private practice located in the Southeast. Send curriculum vitae and references.

Please respond to Box 296. 296C/E

Cardiovascular and thoracic surgeon wanted to join private practice in mid-Atlantic state. Please respond with CV.

Please respond to Box 298. 298C/D

Thoracic/vascular surgeon to join small group private practice in medium-sized New England community. BC/BE in thoracic surgery. Prefer C/E for Cert. Special Competence in Vascular Surgery. Recent medical graduate preferred. University-affiliated hospital. Full partnership in 3 years.

Please respond to Box 301. 301C/F

Cardiothoracic/vascular surgeon wanted to join established practice in Ohio. Recent graduate preferred. Please submit curriculum vitae and three references.

Please respond to Box 302. 302C/F

Thoracic, vascular, and general surgeon to join a busy, all boarded, private practice on eastern Long Island. Should be board certified or eligible. Excellent salary and benefits, leading to a full partnership. Send curriculum vitae.

Please respond to Box 303. 303C/E

Thoracic and vascular surgeon, board certified, to join active (noncardiac) thoracic and vascular practice in northern New Jersey. Competitive salary and benefits. Please respond with curriculum vitae.

Please respond to Box 304. 304C/E

Cardiovascular/thoracic surgeon, BC/BE, to join established private practice. Desirable location on Florida west coast. Expertise in adult cardiac, vascular, and thoracic surgery required. Recent graduate, transplant experience preferred. Please include curriculum vitae and references with response.

Please respond to Box 305. 305C/D

Faculty position: The Division of Thoracic and Cardiovascular Surgery, Southwestern Medical School at Dallas, is seeking an individual with strong clinical background in adult cardiovascular/thoracic surgery along with a commitment to education and research for a position at the assistant professor level. Must be ABTS certified or eligible.

Send complete curriculum vitae to W. Steves Ring, MD, Chairman, Division of Thoracic and Cardiovascular Surgery, UT Southwestern Medical Center, 5323 Harry Hines Blvd, Dallas, TX 75235-9031. An equal opportunity employer. 309D

Group of two cardiothoracic surgeons seeking third associate. Recent graduate preferred. American Board of Thoracic Surgery certification or eligibility is required. Practice consists of adult cardiac surgery at University-affiliated hospital in the New York metropolitan area.

Please respond to Box 310. 310D/F

Cardiovascular surgeon to become second surgeon in new (two year old) adult cardiac program on west coast of Florida.

Submit CV to Manatee Cardiac Surgeons, PA, 201 4th Ave East #2, Bradenton, FL 34208. 312D/F

Cardiothoracic surgeon (BE/BC) to join newly established practice of primarily adult cardiac surgery. Expanding practice is now ready for second surgeon. Excellent opportunity for a recent graduate in the beautiful area of Virginia.

Please respond to Box 313. 313D

Thoracic-vascular surgeon wanted to join busy practice, with outstanding growth potential, in major city along Eastern seaboard. Board certified or eligible. Interested in practice of thoracic-vascular surgery. Cardiac interests not excluded. Excellent compensation and benefits. Send curriculum vitae.

Please respond to Box 314. 314D

Cardiac surgeon wanted to join established private practice in Eastern PA. Primarily adult cardiac. BC/BE required. Please send CV.

Please respond to Box 315. 315D/I

Cardiovascular and thoracic surgeon, board eligible or certified, wanted to join another physician in private practice in Northeast. Immediate availability. Please respond with curriculum vitae.

Please respond to Box 316. 316D/I

Cardiovascular-thoracic surgeon, board certified or eligible wanted for established fast-growing practice in south Texas with modern hospital facilities; primarily adult cardiac surgery, thoracic and vascular. Initial contact should include curriculum vitae, clinical experience, surgical results, and names of references.

Please respond to Box 319. 319D/E

Cardiac surgeon with additional interest and experience in vascular surgery wanted to join large, active group practice in suburban Washington, DC. Prefer recent ABTS-certified surgeon with 2 to 5 years experience.

Please respond to Box 322. 322D/F

Academic Program Director, General Surgery Residency Training Program. MacNeal Hospital, a west suburban Chicago teaching hospital, is seeking an Academic Program Director to lead its General Surgery Residency Training Program. The individual must be board certified in general surgery and a surgical subspecialty. The candidate selected will assume appropriate administrative and teaching responsibilities for the professional and scholarly growth of the department; will also actively participate in patient care and conduct daily rounds with house staff. The ideal candidate has national stature in the field of surgery, with a demonstrated record of excellence in scholarship, clinical service, and academic administration. MacNeal Hospital is a 427-bed facility, accredited to educate 5 General Surgery Residents in an integrated program with the University of Illinois. Academic appointment is required. A competitive salary and benefit package is available.

Candidates should forward a letter of application and CV to Jerry Lee, Vice President, Physician Affairs, MacNeal Hospital, 3249 S Oak Park Ave, Berwyn, IL 60402. 323D/E

Research Associate in Cardiovascular Surgery to work in established, productive, experimental surgery laboratory. Projects in temporary and permanent left ventricular assist systems and related artificial organs. Opportunity to participate in clinical research. Clinical open heart surgery experience essential; research background desirable. Send curriculum vitae.

Please respond to Box 324. 324D

The University of Maryland, Department of Surgery, is seeking two cardiothoracic surgeons at the Assistant Professor level. Successful candidates should have special expertise in pediatric cardiothoracic surgery as well as general thoracic surgery with a major commitment to an academic career.

Please send curriculum vitae and 3 professional references to Joseph S. McLaughlin, MD, Professor and Head, Division of Cardiovascular & Thoracic Surgery, Department of Surgery, University of Maryland, 22 S Greene St, Baltimore, MD 21201. AA/EOE. 325D

Cardiac surgeon: Excellent opportunity to join a very busy adult cardiac surgery practice in Philadelphia. ABTS eligibility required. Recent graduate preferred. Salaried associate leading to partnership.

Send curriculum vitae and three references to John Bell-Thomson, MD, Chairman, Division of Cardiothoracic Surgery, Albert Einstein Medical Center, York & Tabor Roads, Philadelphia, PA 19141. 326D

SITUATIONS WANTED

42-year-old cardiac surgeon, American trained, ABS certified, ABTS eligible. Currently practicing in the Middle East. Has considerable experience in valve surgery. Wishes to relocate to the US but will require a visa. Would consider joining a group or research project.

Please respond to Box 285. 285B/D

Cardiovascular and thoracic surgeon, American, University trained, 36, wishes to relocate. ABS certified, ABTS eligible. Interested in joining a group or initiating a new program.

Please respond to Box 288. 288B/D

Pediatric and adult cardiovascular surgeon, 37, completed fellowship in major university medical center. Extensive clinical and research experience. Interested in academic position or private practice. Curriculum vitae and references upon request.

Please respond to Box 311. 311D/E

Thoracic surgeon—54, American, board certified. General thoracic, peripheral vascular, endoscopy, pacemakers. Twenty years in community practice; wish to relocate. Good communicator. Prefer East, Midwest. CV available.

Please respond to Box 317. 317D

Cardiothoracic surgeon, 34, American, NE university training, 10 months private practice experience, wishes to relocate. ABS, ABTS eligible. Experienced in adult coronary reoperations, ventricular assists, and electrophysiologic surgery. Seeks practice in Southwest/Southeast. Curriculum vitae and references upon request.

Please respond to Box 318. 313D/G

Future resident in Cardiothoracic Surgery with fellowship experience desires operative training and/or research experience in pediatric cardiac, heart/lung transplantation/assist devices, and/or electrophysiological surgery.

Please respond to Box 320. 320D

Cardiovascular and thoracic surgeon, 37, American, graduate July 1990—BE, ABS, ABTS, extensive open heart and noncardiac thoracic experience. Excellent general surgery training, strong peripheral vascular experience.

Please respond to Box 321. 321D

Rome graduate, USA-trained, NYH-Cornell cardiothoracic fellow, would consider joining research project for 2 years.

Please respond to Iolanda Russo, 420 East 70, 5A, New York, NY 10021. 328D

FELLOWSHIPS

Fellowship in cardiovascular surgery for 1 year available January 1 or July 1 with group doing approximately 1,000 cardiac procedures per year and large volume of vascular procedures. ABS eligible or certified, California medical license required or readily obtainable. Ideal for a person between general surgery residency and thoracic surgery residency.

Please respond with curriculum vitae to Bradley J. Harlan, MD, 5301 F Street, Suite 312, Sacramento, CA 95819. 222K/D

Fellowship in pediatric cardiovascular surgery available July 1990. Experience includes surgery for hypoplastic left heart syndrome and arterial switch. A large, well-organized collection of heart specimens available for review. Illinois license or equivalent required. Salary and benefits in the high $40's.

Please respond to Division of Pediatric Cardiac Surgery, The Heart Institute for Children, Christ Hospital and Medical Center, 4440 W 95th St, Oak Lawn, IL 60453; or telephone (312) 857-3029. 253L/D

Postresidency clinical fellowship available in cardiopulmonary surgery with group performing over 1,000 cardiac procedures per year. Up to 25% research opportunity option available. Complete training in cardiopulmonary surgery required. US graduates must be eligible for Oregon license. Foreign medical graduates must be eligible for J-1 or H-1 visa. Fellowship for 1 to 2 years beginning July 1990 or January 1991.

Please send CV to Albert Starr, MD, 9155 SW Barnes Rd #240, Portland, OR 97225. 287B/D

Cardiopulmonary transplant fellowship. Available July 1, 1991, to June 30, 1992. Active transplant program offers surgeon opportunity to assist in donor procurement, operations, pre- and postop management of patients, as well as recipient evaluation. Position is appropriate for PGY III or higher or for surgeon who has completed thoracic surgery.

Submit complete curriculum vitae to John C. Baldwin, MD, Professor and Chief, Cardiothoracic Surgery, Yale University School of Medicine, 333 Cedar Street, PO Box 3333, New Haven, CT 06510. Yale University is an affirmative action/equal opportunity employer. 327D

Physician Assistants

2 positions open for physicians assistants with 6 man Cardiothoracic surgery group. Large volume cardiac and thoracic surgery program. Excellent salary. Please contact:

Dr. Thomas Z. Lajos
Buffalo Heart Surgical Associates
56 Linwood Ave.
Buffalo, NY 14209
Tel. (716) 845-2249

CARDIOVASCULAR SURGEON NEEDED FOR SAGINAW, MICHIGAN

Two man cardiovascular surgical group, with an active, expanding practice in Eastern Mid-Michigan, has an immediate opening for a cardiovascular surgeon, Board Certified/Board Eligible.

Saginaw, Michigan is the gateway to beautiful northern Michigan and serves as the focal point for a fourteen county health planning area, which encompasses close to 1,000,000 people.

For further information about this exceptional private practice opportunity, please send a CV to:

Ms. Donna Bowden
East Michigan Cardiovascular Surgery, P.C.
100 Harrow Lane
Saginaw, MI 48603

CARDIAC SURGERY FELLOWSHIPS & ASSOCIATE POSITIONS IN CARDIAC SURGERY

The Cleveland Clinic Foundation's Department of Cardiovascular Surgery performs 3000 open heart operations annually with an increasing proportion of either complex procedures or reoperation. Active sections exist in all facets of adult cardiac surgery including: reparative valvular procedures, electrophysiological surgery, cardiac transplantation, and circulatory support. Both clinical and experimental research opportunities exist.

FELLOWSHIP
A 1-2 year fellowship is available for individuals interested in board equivalent training in cardiac surgery. This program covers all aspects of adult cardiac surgery including intensive care unit experience.

CLINICAL ASSOCIATE
Individuals who have completed their training in Cardiac Surgery who are interested in an additional 1-2 year period of experience in adult cardiac surgery are eligible for the Clinical Associate Position.

Both positions are associated with competitive salaries, opportunities for attendance at national meetings and allotted vacation times. For information please contact: Robert W. Stewart, M.D.

The Cleveland Clinic Foundation
One Clinic Center
9500 Euclid Avenue
Cleveland, OH 44195-5066

or call: (216) 444-6708

S&W SCOTT&WHITE

CARDIOVASCULAR SURGEON NEEDED

Scott & White, a multi-specialty Clinic of over 300 physicians in Temple, Texas, needs a cardiovascular and thoracic surgeon to join its 3-Surgeon division.

Temple is located in Central Texas between Austin and Dallas. Population: 50,000.

Excellent benefit package. Affiliated with Texas A&M University College of Medicine.

For details, contact: F. L. Korompai, M.D., Scott & White Clinic, 2401 South 31st Street, Temple, Texas, 76508, 817-774-4910.

Cardiac Surgery Fellowship

One year fellowship available in the Division of Cardiac Surgery, University of Medicine & Dentistry of New Jersey-Robert Wood Johnson Medical School, beginning July, 1990. Rapidly expanding University Cardiac Surgery Service has opportunity for fellow to assist in operating room and help care for post-operative patients. Active research laboratories can provide opportunities for clinical and/or basic research. Situation is ideal for individual who has completed General Surgery and plans to continue into Cardiac Surgery Training Program.

Please contact: **Alan J. Spotnitz, M.D., UMDNJ-Robert Wood Johnson Medical School, One Robert Wood Johnson Place, CN 19, (ATS), New Brunswick, NJ 08903-0019.** The UMDNJ is an Affirmative Action/Equal Employment Opportunity Employer M/F/H/V and a member of the University Health System of New Jersey.

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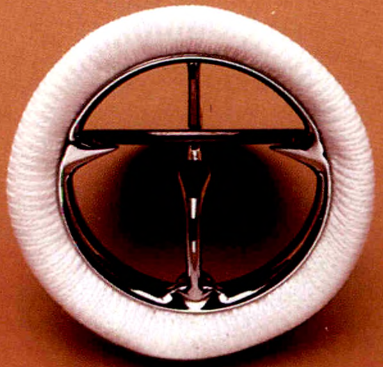

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For a complete listing of indications and usage, please consult Brief Summary of Prescribing Information for ZINACEF on adjacent page.

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Zinacef*
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Zinacef*
(cefuroxime sodium injection)

BRIEF SUMMARY

The following is a brief summary only. Before prescribing, see complete prescribing information in Zinacef* product labeling.

INDICATIONS AND USAGE: Zinacef* is indicated for the treatment of patients with infections caused by susceptible strains of the designated organisms in the following diseases

1. **Lower Respiratory Tract Infections**, including pneumonia, caused by *Streptococcus pneumoniae* (formerly *Diplococcus pneumoniae*), *Haemophilus influenzae* (including ampicillin-resistant strains), *Klebsiella* spp, *Staphylococcus aureus* (penicillinase- and non-penicillinase-producing), *Streptococcus pyogenes* and *Escherichia coli*
2. **Urinary Tract Infections** caused by *E coli* and *Klebsiella* spp
3. **Skin and Skin Structure Infections** caused by *S aureus* (penicillinase- and non-penicillinase-producing), *S pyogenes*, *E coli*, *Klebsiella* spp, and *Enterobacter* spp
4. **Septicemia** caused by *S aureus* (penicillinase- and non-penicillinase-producing), *S pneumoniae*, *E coli*, *H influenzae* (including ampicillin-resistant strains), and *Klebsiella* spp
5. **Meningitis** caused by *S pneumoniae*, *H influenzae* (including ampicillin-resistant strains), *Neisseria meningitidis*, and *S aureus* (penicillinase- and non-penicillinase-producing)
6. **Gonorrhea:** Uncomplicated and disseminated gonococcal infections due to *Neisseria gonorrhoeae* (penicillinase- and non-penicillinase-producing strains) in both males and females.
7. **Bone and Joint Infections** caused by *S aureus* (including penicillinase- and non-penicillinase-producing strains)

Clinical microbiological studies in skin and skin structure infections frequently reveal the growth of susceptible strains of both aerobic and anaerobic organisms. Zinacef has been used successfully in these mixed infections in which several organisms have been isolated. Appropriate cultures and susceptibility studies should be performed to determine the susceptibility of the causative organisms to Zinacef

Therapy may be started while awaiting the results of these studies, however, once these results become available, the antibiotic treatment should be adjusted accordingly. In certain cases of confirmed or suspected gram-positive or gram-negative sepsis or in patients with other serious infections in which the causative organism has not been identified, Zinacef may be used concomitantly with an aminoglycoside (see PRECAUTIONS). The recommended doses of both antibiotics may be given depending on the severity of the infection and the patient's condition

Prevention: The preoperative prophylactic administration of Zinacef may prevent the growth of susceptible disease-causing bacteria and thereby may reduce the incidence of certain postoperative infections in patients undergoing surgical procedures (eg, vaginal hysterectomy) that are classified as clean-contaminated or potentially contaminated procedures. Effective prophylactic use of antibiotics in surgery depends on the time of administration. Zinacef should usually be given one-half to one hour before the operation to allow sufficient time to achieve effective antibiotic concentrations in the wound tissues during the procedure. The dose should be repeated intraoperatively if the surgical procedure is lengthy.

Prophylactic administration is usually not required after the surgical procedure ends and should be stopped within 24 hours. In the majority of surgical procedures, continuing prophylactic administration of any antibiotic does not reduce the incidence of subsequent infections but will increase the possibility of adverse reactions and the development of bacterial resistance

The perioperative use of Zinacef has also been effective during open heart surgery for surgical patients in whom infections at the operative site would present a serious risk. For these patients it is recommended that Zinacef therapy be continued for at least 48 hours after the surgical procedure ends. If an infection is present, specimens for culture should be obtained for the identification of the causative organism and appropriate antimicrobial therapy should be instituted

CONTRAINDICATIONS: Zinacef* is contraindicated in patients with known allergy to the cephalosporin group of antibiotics

WARNINGS: BEFORE THERAPY WITH ZINACEF* IS INSTITUTED, CAREFUL INQUIRY SHOULD BE MADE TO DETERMINE WHETHER THE PATIENT HAS HAD PREVIOUS HYPERSENSITIVITY REACTIONS TO CEPHALOSPORINS, PENICILLINS, OR OTHER DRUGS. THIS PRODUCT SHOULD BE GIVEN CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS. ANTIBIOTICS SHOULD BE ADMINISTERED WITH CAUTION TO ANY PATIENT WHO HAS DEMONSTRATED SOME FORM OF ALLERGY, PARTICULARLY TO DRUGS. IF AN ALLERGIC REACTION TO ZINACEF OCCURS, DISCONTINUE THE DRUG. SERIOUS ACUTE HYPERSENSITIVITY REACTIONS MAY REQUIRE EPINEPHRINE AND OTHER EMERGENCY MEASURES

Pseudomembranous colitis has been reported with the use of cephalosporins (and other broad-spectrum antibiotics); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with antibiotic use.

Treatment with broad-spectrum antibiotics alters normal flora of the colon and may permit overgrowth of clostridia. Studies indicate a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis. Cholestyramine and colestipol resins have been shown to bind the toxin *in vitro*

Mild cases of colitis may respond to drug discontinuation alone. Moderate to severe cases should be managed with fluid, electrolyte, and protein supplementation as indicated

When the colitis is not relieved by drug discontinuation or when it is severe, oral vancomycin is the treatment of choice for antibiotic-associated pseudomembranous colitis produced by *C difficile*. Other causes of colitis should also be considered

PRECAUTIONS: Although Zinacef* rarely produces alterations in kidney function, evaluation of renal status during therapy is recommended, especially in seriously ill patients receiving the maximum doses. Cephalosporins should be given with caution to patients receiving concurrent treatment with potent diuretics as these regimens are suspected of adversely affecting renal function

The total daily dose of Zinacef should be reduced in patients with transient or persistent renal insufficiency (see DOSAGE AND ADMINISTRATION), because high and prolonged serum antibiotic concentrations can occur in such individuals from usual doses

As with other antibiotics, prolonged use of Zinacef may result in overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis

Nephrotoxicity has been reported following concomitant administration of aminoglycoside antibiotics and cephalosporins

Drug/Laboratory Test Interactions: A false-positive reaction for glucose in the urine may occur with copper reduction tests (Benedict's or Fehling's solution or with Clinitest* tablets), but not with enzyme-based tests for glycosuria (eg, Tes-Tape*). As a false-negative result may occur in the ferricyanide test, it is recommended that either the glucose oxidase or hexokinase method be used to determine blood plasma glucose levels in patients receiving Zinacef

Cefuroxime does not interfere with the assay of serum and urine creatinine by the alkaline picrate method

Carcinogenesis, Mutagenesis, Impairment of Fertility: Although no long-term studies in animals have been performed to evaluate carcinogenic potential, no mutagenic potential of cefuroxime was found in standard laboratory tests

Reproductive studies revealed no impairment of fertility in animals

Pregnancy: ***Teratogenic Effects: Pregnancy Category B:*** Reproduction studies have been performed in mice and rabbits at doses up to 60 times the human dose and have revealed no evidence of impaired fertility or harm to the fetus due to cefuroxime. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed

Nursing Mothers: Since Zinacef is excreted in human milk, caution should be exercised when Zinacef is administered to a nursing woman

Pediatric Use: Safety and effectiveness in children below 3 months of age have not been established. Accumulation of other members of the cephalosporin class in newborn infants (with resulting prolongation of drug half-life) has been reported

ADVERSE REACTIONS: Zinacef* is generally well tolerated. The most common adverse effects have been local reactions following intravenous administration. Other adverse reactions have been encountered only rarely

Local Reactions: Thrombophlebitis has occurred with intravenous administration in 1 in 60 patients

Gastrointestinal: Gastrointestinal symptoms occurred in 1 in 150 patients and included diarrhea (1 in 220 patients) and nausea (1 in 440 patients). Onset of pseudomembranous colitis symptoms may occur during or after antibiotic treatment (see WARNINGS)

Hypersensitivity Reactions: Hypersensitivity reactions have been reported in less than 1% of the patients treated with Zinacef and include rash (1 in 125). Pruritus, urticaria, and positive Coombs' test each occurred in less than 1 in 250 patients and, as with other cephalosporins, rare cases of anaphylaxis, erythema multiforme, and Stevens-Johnson syndrome have occurred

Blood: A decrease in hemoglobin and hematocrit has been observed in 1 in 10 patients and transient eosinophilia in 1 in 14 patients. Less common reactions seen were transient neutropenia (less than 1 in 100 patients) and leukopenia (1 in 750 patients). A similar pattern and incidence were seen with other cephalosporins used in controlled studies

Hepatic: Transient rise in SGOT and SGPT (1 in 25 patients), alkaline phosphatase (1 in 50 patients), LDH (1 in 75 patients), and bilirubin (1 in 500 patients) levels has been noted

Kidney: Elevations in serum creatinine and/or blood urea nitrogen and a decreased creatinine clearance have been observed, but their relationship to cefuroxime is unknown

In addition to the adverse reactions listed above that have been observed in patients treated with cefuroxime, the following adverse reactions and altered laboratory tests have been reported for cephalosporin-class antibiotics

Adverse Reactions: Toxic epidermal necrolysis, fever, vomiting, abdominal pain, colitis, vaginitis including vaginal candidiasis, toxic nephropathy, hepatic dysfunction including cholestasis, aplastic anemia, hemolytic anemia, hemorrhage

Several cephalosporins have been implicated in triggering seizures, particularly in patients with renal impairment when the dosage was not reduced (see DOSAGE AND ADMINISTRATION). If seizures associated with drug therapy should occur, the drug should be discontinued. Anticonvulsant therapy can be given if clinically indicated

Altered Laboratory Tests: Prolonged prothrombin time, pancytopenia, agranulocytosis, thrombocytopenia

DOSAGE AND ADMINISTRATION: (See complete prescribing information in Zinacef* product labeling.)

Impaired Renal Function: A reduced dosage must be employed when renal function is impaired. Dosage should be determined by the degree of renal impairment and the susceptibility of the causative organism (see Table 1 in product package insert)

HOW SUPPLIED: Zinacef* in the dry state should be stored between 15° and 30°C (59° and 86°F) and protected from light. Zinacef is a dry, white to off-white powder supplied in vials and infusion packs as follows

NDC 0173-0352-31	750-mg* Vial (Tray of 25)
NDC 0173-0354-35	1.5-g* Vial (Tray of 25)
NDC 0173-0353-32	750-mg* Infusion Pack (Tray of 10)
NDC 0173-0356-32	1.5-g* Infusion Pack (Tray of 10)
NDC 0173-0400-00	7.5-g* Pharmacy Bulk Package (Tray of 6)

Zinacef frozen as a premixed solution of cefuroxime sodium should not be stored above -20°C. Zinacef is supplied frozen in 50-mL, single-dose, plastic containers as follows.

NDC 0173-0424-00 750-mg* Plastic Container (Carton of 24)
NDC 0173-0425-00 1.5-g* Plastic Container (Carton of 24)

*Equivalent to cefuroxime

October 1989
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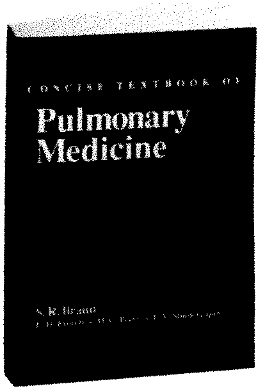

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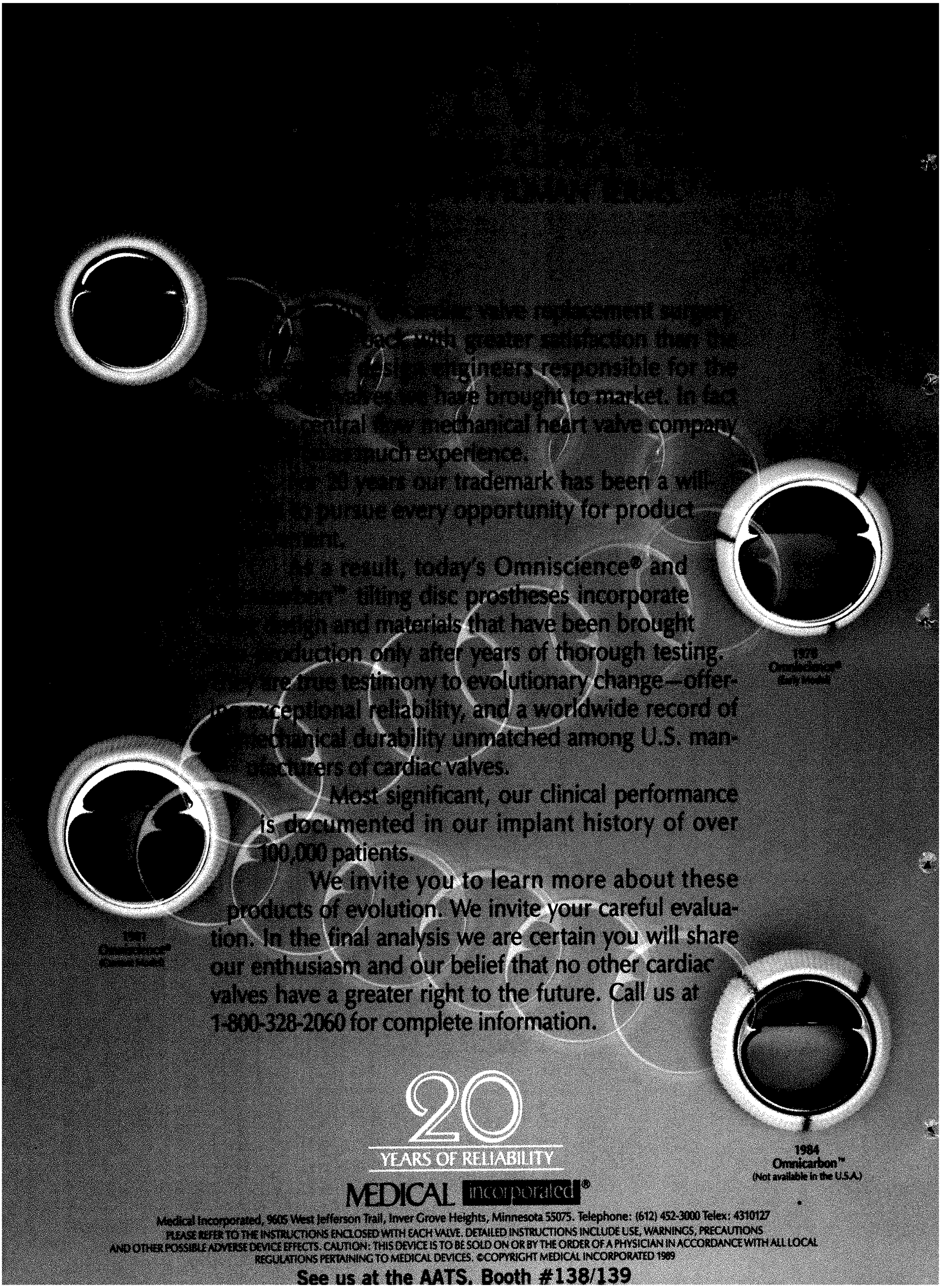

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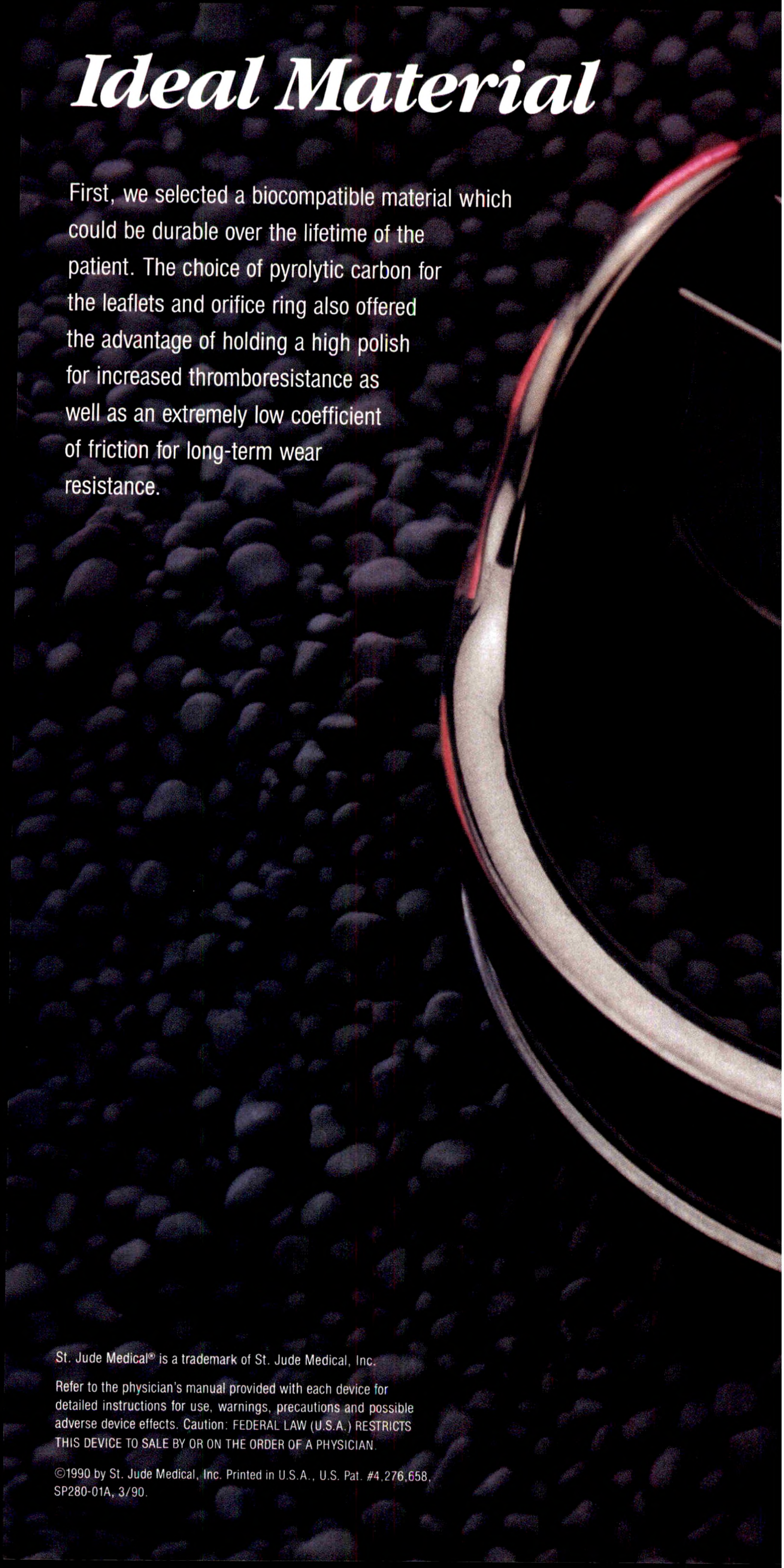

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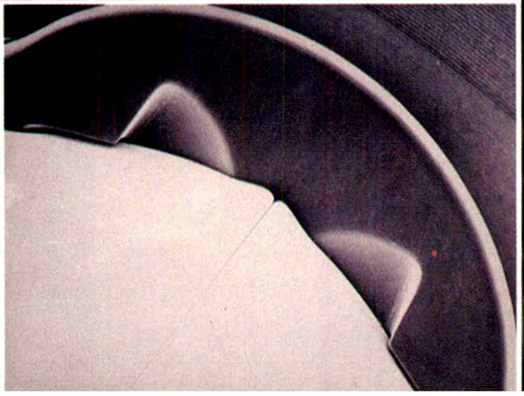

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The Carpentier-Edwards bioprosthesis is intended for use in patients whose aortic or mitral valvular disease is sufficiently advanced to warrant replacement of their natural valve with a prosthetic one. It is also intended for use in patients with a previously implanted aortic or mitral valve prosthesis which is no longer functioning adequately and requires replacement. In the latter case, the previously implanted prosthesis is surgically excised and replaced by the replacement prosthesis. **Warnings:** For Single Patient Use Only In the presence of conditions affecting calcium metabolism or when calcium-containing chronic drug therapies are used, the use of a mechanical prosthesis as an alternative should be considered. This is also true in patients under 20 years of age, in patients on a high calcium diet, in the presence of severe systemic hypertension, and in patients who are on maintenance hemodialysis. The decision to use a tissue valve must ultimately be made by the physician on an individual basis after a careful evaluation of the short- and long-term risks and benefits to the patient are made, and alternative methods of treatment are considered. Overall durability, especially long-term, has not been established for bioprostheses. Serious complications, sometimes leading to replacement of the valve and/or death, may be associated with the use of prosthetic valves (see Complications). A full explanation of the benefits and risks should be given to each prospective patient before surgery. **Note:** Bioprostheses should be used with caution in the presence of severe systemic hypertension or when the anticipated patient longevity is longer than the known longevity of the prosthesis. Careful and continuous medical follow-up (at least by an annual visit to the physician) is advised so that valve-related complications, particularly those related to material failure, can be diagnosed and properly managed.Recipients of prosthetic heart valves who are undergoing dental procedures should receive prophylactic antibiotic therapy to minimize the possibility of prosthetic infection. Bioprosthetic heart valve recipients should be maintained on anticoagulant therapy, except where contraindicated, during the initial healing stages after implantation, approximately 2 to 3 months. Anticoagulants should then be discontinued over a period of 10 days, except in those patients for whom indefinite anticoagulant protection is indicated, i.e., in the absence of sinus rhythm and in patients with a dilated left atrium, calcification of the atrial wall or history of previous atrial thrombosis. The appropriate anticoagulation therapy must be determined by the physician on an individual basis. **Adequate rinsing with physiological saline, as described in the Technique section, is mandatory before implantation to reduce the glutaraldehyde concentration. No other solutions, drugs, chemicals, antibiotics, etc. should ever be added to the glutaraldehyde or rinse solutions, as irreparable damage to the leaflet tissue, which may not be apparent under visual inspection, may result. The Carpentier-Edwards bioprostheses must be kept moist at all times. Drying out will cause irreversible damage to the leaflet tissue. To prevent drying out during implantation, the valves should be irrigated periodically on both sides with sterile physiological saline.** Passing diagnostic catheters and transvenous pacing leads across a Carpentier-Edwards bioprosthesis could damage the valve. Consequently, it is recommended that these procedures be avoided. Both the valve and glutaraldehyde storage solution are **sterile.** The outside of the jar is **not sterile** and must **not** be placed in the sterile field. Furthermore, the valve and container should **never** be subjected to sterilization procedures involving ethylene oxide, propylene oxide, steam, or irradiation. Storage between 10°C and 25°C is recommended; however, care should be exercised to avoid freezing or extreme heat, which may damage the valvular tissue. **Any valve that has been frozen or exposed to temperatures greater than 60°C should not be used for human implantation.** Glutaraldehyde may cause irritation of the skin, eyes, nose, and throat, and may also cause skin sensitization. Avoid prolonged or repeated contact or prolonged breathing of the vapor. Use only with adequate ventilation. In the event of contact, immediately flush the affected area with water. In the event of contact with eyes, seek medical attention. **Surgical Precautions: 1.** When choosing a valve for a given patient, the size, age, and physical condition of the patient in relation to the size of the prosthesis must be taken into consideration to minimize the possibility of obtaining a suboptimal hemodynamic result. The selection of a valve, however, must ultimately be made by the physician on an individual basis after carefully weighing all of the risks and benefits to the patient. **2.** Due to the relative flexibility of the frame, care must be exercised to avoid the use of too large a prosthesis to prevent folding or extreme deformation of the stent that may render the valve incompetent. Oversizing may create highly localized mechanical stresses resulting in tissue failure in the form of detachment of the leaflets or porcine aortic wall from the valve stent posts. **3.** Left ventricular perforation or ulceration has been reported. Both open and closed chest cardiac massage in patients with an open strut mitral prosthesis increases the risk of ventricular perforation. Additionally, lifting, flexing, and manual compression of the distended heart after discontinuation of bypass may cause a commissure strut to be pushed through the endocardium, producing a dissecting hematoma with subsequent uncontrolled hemorrhage. **4.** The spacing of the sutures in the remnant of the valvular orifice and the prosthesis suture ring must be carefully matched to avoid folding of the leaflets or extreme distortion of the orifice. Edwards CVS Division has received reports in which individual mattress sutures, spanning a distance of 10 to 15mm, produced a purse-string effect causing compression of the valve orifice. **5.** When using interrupted sutures, it is important to cut the sutures close to the knots and to ensure that exposed suture tails will not come into contact with the leaflet tissue. Cases have been reported in which bioprostheses developed severe regurgitation and had to be replaced as a result of wear due to contact with sutures. **6.** Unlike rigid mechanical valves, the stent wall is soft and will not resist needle penetration. Accordingly, extreme care must be exercised when placing sutures through the sewing margin to avoid penetration of the side wall of the stent and possible laceration of the leaflet tissue. **7.** As with all prostheses that have open cages, free struts, or commissure supports, care must be exercised, particularly in mitral replacement, to avoid looping or catching a suture around the free legs which would interfere with proper valvular function. **8.** The stent of the bioprosthesis is asymmetrical to conform to the natural shape of the porcine aortic valve. Consequently, the commissure supports are not equidistantly spaced. The contrasting suture markers in the sewing ring of the mitral valve denote the greatest intercommissural distance. These two commissure supports should be oriented so as to straddle the ventricular outflow tract to avoid murmurs due to blood flow turbulence. **Note:** The contrasting suture material should not be removed from the prosthesis. **9.** In the aortic position, the struts should correspond to the remnants of the natural commissures so as not to obstruct the coronary ostia. **10.** A serial number tag is attached to the sewing ring of each valve by a suture. This serial number should be checked against the number on the jar; if any difference is noted, the valve should be returned unused. This tag should not be detached from the valve until implant is imminent. Care should be exercised to avoid cutting or tearing the suture ring cloth during removal. **11.** Gentle handling is required for all implantable devices. If the valve is dropped, damaged, or mishandled in any way, it must not be used for human implantation. **Complications:** As with all prosthetic heart valves, serious complications, sometimes leading to death, may be associated with the use of tissue valves. In addition, complications due to individual patient reaction to an implanted device, or to physical or chemical changes in the components, particularly those of biological origin, may occur at varying intervals (hours or days) necessitating reoperation and replacement of the prosthetic device. Complications associated with the use of Carpentier-Edwards bioprostheses include stenosis, regurgitation through an incompetent valve, perivalvular leak, endocarditis, hemolysis, thromboembolism, thrombotic obstruction, bleeding diatheses related to the use of anticoagulant therapy, ventricular perforation by stent posts, looped sutures, and malfunctions of the valve due to distortion at implant or physical or chemical deterioration of valve components. Types of tissue deterioration include infection, calcification, thickening, perforation, suture abrasion, instrument trauma, and leaflet or aortic wall detachment from the valve stent posts. These complications may present clinically as abnormal heart murmur, shortness of breath, exercise intolerance, dyspnea, orthopnea, anemia, fever, arrythmia, hemorrhage, transient ischemic attack, stroke, paralysis, low cardiac output, pulmonary edema, congestive heart failure, cardiac failure, and myocardial infarct. **Note:** Based on reports in the literature on tissue valves, there appears to be an increased incidence of leaflet calcification in patients under the age of 20. In this regard, animal research studies show that a high systemic calcium level can lead to early calcification. Furthermore, at least one published report describes an apparent causal relationship between the consumption of daily calcium supplements and early leaflet calcification in an adult. When feasible, repeated intravenous injections containing calcium should be avoided during the postoperative period; and excessive milk or dairy product consumption should be avoided in children. There are no clinical data presently available demonstrating increased resistance of Carpentier-Edwards bioprostheses to calcification as compared to other commercially available porcine valves. **Caution:** Federal (USA) law restricts this device to sale by or on the order of a physician. Baxter International Inc. is the owner of the following US patent which covers the products described herein: 4,106,129. Patent numbers in foreign countries supplied upon request. See package insert accompanying product for a full discussion of warnings, contraindications, complications and prescribing information. Carpentier-Edwards® is a registered trademark of Baxter International Inc.

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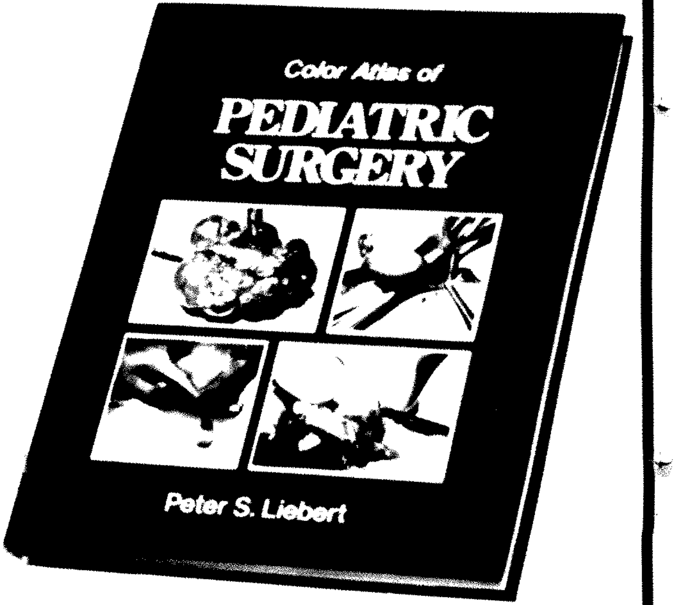

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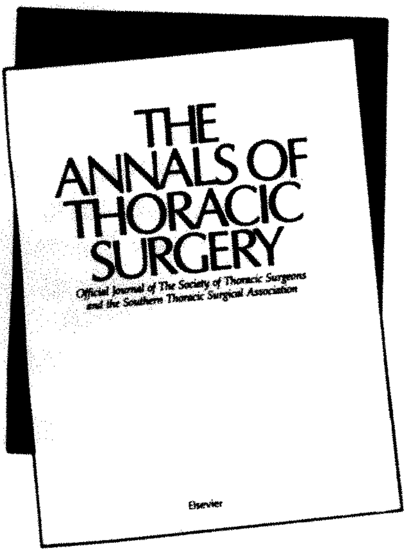

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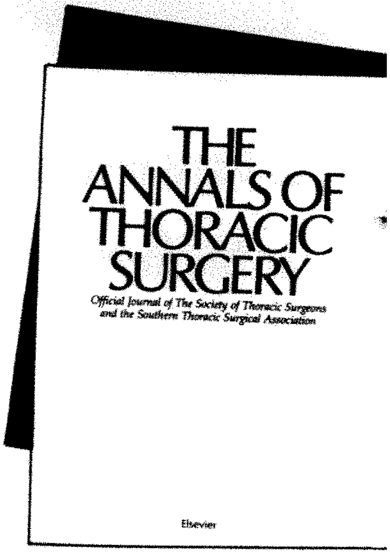

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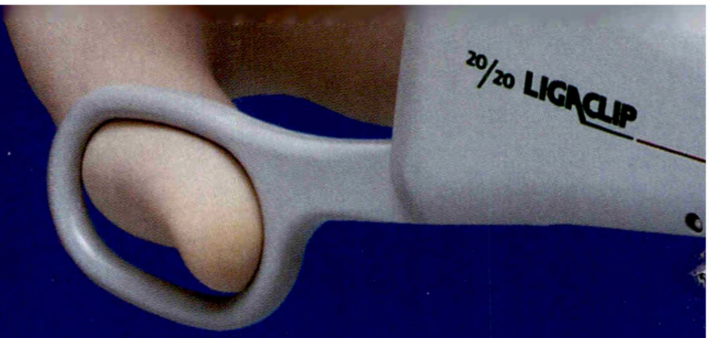

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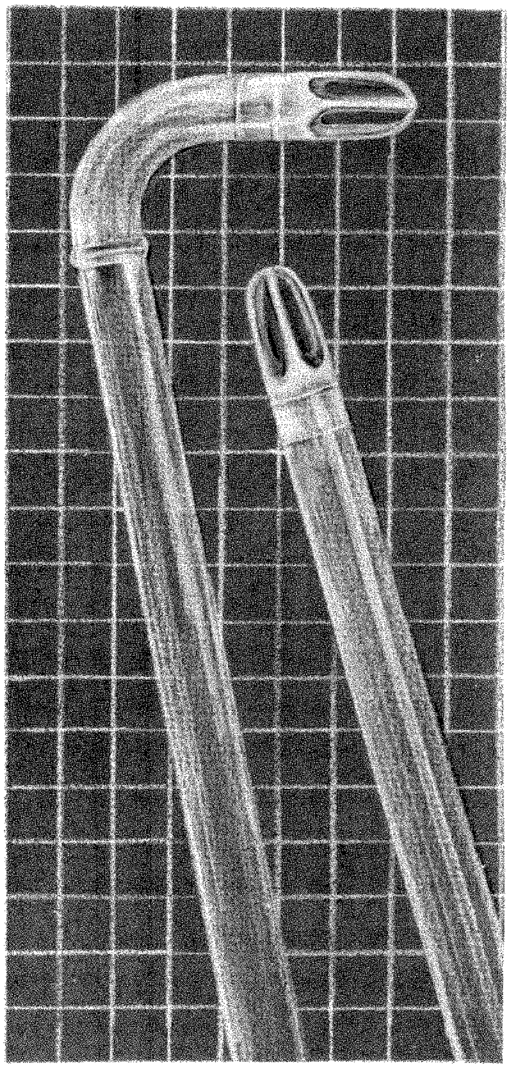

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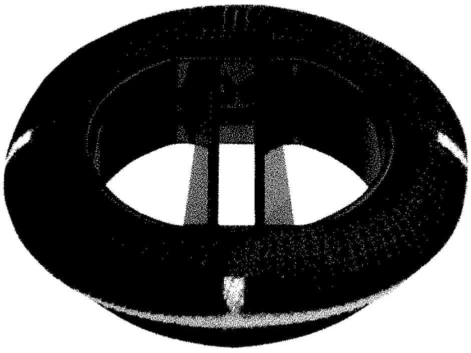

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Aortic Valve Decalcification: History Repeated With a New Result

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Decalcification of stenotic aortic valves was one of the original cardiac surgical procedures. It fell into disuse because of an exceptionally high incidence of early restenosis and because prosthetic valves that produced more predictable early and superior long-term results became available. The operation was revived when ultrasonic debridement was reported by Brown and Davies [1] in 1972. They modified a dental instrument and applied it to trileaflet valves with calcific aortic stenosis. The rationale for this approach was that more complete removal of the calcium with less tissue trauma would reduce the incidence of cusp perforation and restenosis. The concept lay dormant for 15 years until a resurgence of interest in debridement of calcific aortic stenosis using ultrasonic debridement was triggered by reports from Mindich and associates [2] and Freeman and co-workers [3]. They demonstrated that aortic stenosis could be effectively relieved without causing aortic insufficiency. Although there were no long-term results, there was reason to hope that the results from ultrasonic debridement of the aortic cusps would be superior to those obtained with mechanical debridement. As the imperfection of all valve substitutes and the advantages of reconstruction of atrioventricular valves became increasingly obvious, surgeons embraced this modality. Most investigators applied the new technology cautiously, selecting older patients with limited life expectancy and younger patients undergoing other cardiac surgical procedures, to relieve aortic valve gradients that did not justify valve replacement. The objective of decalcification in the latter group was to slow the progression of the stenosis and thus postpone or eliminate the necessity for a subsequent procedure on the aortic valve.

See also page 746.

There have been few previous reports of late results in patients undergoing ultrasonic debridement. We are indebted to Craver [4] for bringing to our attention the unexpected results of this clinical trial. Although most investigators foresaw a significant incidence of restenosis, none suspected there would be an early occurrence of severe and progressive aortic insufficiency. Craver found that 3 of 10 patients required reoperation for aortic insufficiency. We have had a similar experience. At The Cleveland Clinic, 5 of 27 patients (19%) required reoperation with a mean follow-up of 26 months. At the Mayo Clinic, 6 of 47 patients (14%) required reoperation at a mean follow-up of 9 months. In addition to the incidence of aortic insufficiency requiring operation, the Mayo Clinic surgeons found progression to severe aortic regurgitation in 26% and moderate insufficiency in 37% of the patients. None of these patients had aortic insufficiency in the immediate postoperative period.

At the Mayo Clinic and The Cleveland Clinic, pathological findings were similar to those reported by Craver. We found leaflet retraction and loss of central coaptation causing central regurgitation. Microscopic examination showed residual fibrosis and calcification with proliferative granulation tissue and early fibrosis thought to be secondary to the healing response. There was, however, a major difference in valve pathology between the two institutions. Valves from The Cleveland Clinic were fibrotic but the individual layers of the valve (fibrosa and ventricularis) with their elastic tissue were still evident. Foci of calcification, either residual or recurrent, were frequent. In contrast, valves from the Mayo Clinic were more severely fibrotic, the leaflets were smaller suggesting retraction, the ventricularis and fibrosa were difficult to identify, and the normal elastic layers were not seen, apparently having been replaced by fibrous tissue. Foci of calcification in these valves were uncommon. The reason for this difference is unknown but may be explained by variations in power settings during ultrasonic debridement, aggressiveness of debridement, or differences in postoperative interval.

It is clear from these results that the use of ultrasonic debridement of the aortic valve should be discontinued as a clinical trial. However, as with many failed procedures, several questions have been raised that will require further investigation. It is assumed that the energy imparted by the debridement causes injury to the tissue; however, none of the institutions used a constant power setting and there are no data available regarding how much trauma valve tissue will endure without retracting. Is it, in fact, possible to remove calcium from the leaflets without causing sufficient injury to the tissue to result in restenosis or insufficiency? If so, how much energy is safe? Unfortunately, this problem will be difficult to investigate because no experimental model is available.

What should be the strategy for handling a minimally stenotic aortic valve at the time of another cardiac proce-

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dure? Currently, there is not enough information regarding the rate of progression of aortic stenosis to enable us to make informed decisions regarding the best course of treatment. Further, it is not known whether removal of calcium will impede or accelerate the rate of progression of aortic stenosis. It would appear that until we have a better understanding of the rate of progression of aortic stenosis and the potential for causing tissue injury, clinical investigation of this type is unwarranted.

The experience has not been for naught. The introduction of ultrasonic debridement provides the cardiac surgeon with a new tool. This device has been most useful in removing heavy calcification from the mitral annulus during valve replacement. With this approach, it is possible to remove deeply embedded calcium with no apparent injury to surrounding soft tissue, thus providing a well-prepared annulus and reducing the potential for atrioventricular rupture and paravalvar leaks.

The publication of these results may seal the fate of ultrasonic debridement of the aortic valve; however, the search for improved results in the operative management of aortic valve disease continues along several imaginative avenues. Preservation of native tissue by valve repair continues to be an attractive concept and new techniques to relieve aortic insufficiency continue to emerge. Cryopreservation of aortic homografts has resurrected this concept but the associated problems of procurement, supply, and implantation will limit this technique. Stentless porcine xenografts and advanced mounting techniques for pericardium hold substantial promise.

We must remember the lessons of the past and recognize that new technology and new application demand reinterpretation of old data and conclusions. Four examples have been given of discarded ideas that are being reapplied and showing substantial potential. In our discouragement with ultrasonic debridement we must not lose sight of the ultimate goal of restoring normal structure and function to the valve.

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Operation for Stage IIIa Small Cell Lung Cancer?

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In this issue of *The Annals* Salzer and associates [1] describe their 29-year surgical experience with 25 cases of small cell lung cancer with ipsilateral mediastinal lymph node involvement (N2) that were treated with operation with or without chemotherapy and radiotherapy. There were 14 patients treated with chemotherapy, radiotherapy, and operation between 1978 and 1989 who had a median survival of 32 months and a projected 5-year survival rate of 47%. Their survival was superior to that of 11 patients treated between 1970 and 1977 with resection alone, resection plus chemotherapy, or resection plus radiotherapy. None of these 11 patients lived longer than 20 months. Salzer and associates conclude that "the results are so encouraging that we can no longer advocate general refusal of radical lung resection for small cell lung carcinoma [SCLC] in the N2 stage . . ."

Most recent combined modality series employing resection in N2 SCLC patients have had rather poor results [2–5]. The results in 14 patients in this series appear better than in other series. Thus, we must examine this group in detail. The staging procedures are not defined by the authors and must have varied considerably over this 29-year period. The definition of N2 disease also changed over this period, and it is not clear whether Salzer and associates are restricting their cases to ipsilateral mediastinal nodes. Three of the 14 patients in the "comprehensive" series had clinical stage I or II disease and were found to have N2 disease only at initial operation. Not surprisingly, these 3 patients did better than the remaining 11 clinical stage IIIa patients.

See also pages 692 and 759.

Salzer and associates misquote the literature in making their case for the role of operation, underestimating the results obtained with current chemotherapy plus radiotherapy. They state that chemotherapy and radiation therapy alone cannot produce 5-year survival. This is clearly not true. Several recent series [6–9] have shown 5-year survival rates of 15% to 40% after chemotherapy and radiation therapy for *all* limited stage patients, the majority of whom have clinical stage IIIb disease (not IIIa disease as Salzer and associates suggest). These survival statistics were achieved initially with concurrent chemotherapy (cyclophosphamide, CCNU, methotrexate)-radiotherapy combinations developed in the 1970s [6]. More recent chemotherapy regimens (etoposide and cisplatin) given with concurrent radiotherapy may produce superior results, even in a cooperative group setting [7]. The use of this two-drug regimen given with twice-daily radiotherapy has produced results equivalent to those of Salzer and associates in two series from the University of Pennsylvania and the National Cancer Institute [8, 9].

Many series have shown excellent results of operation followed by chemotherapy in clinical stage I and II SCLC. However, the excellent results obtained in recent series [6–9] of stage III SCLC patients without operation means that the addition of operation to these regimens should be proved to be beneficial in a prospective randomized series.

Because the series of Salzer and associates and other surgical series show excellent survival in selected limited stage patients, I believe the role of operation in patients with stage IIIa SCLC remains undefined. Fortunately, an international consortium including the Lung Cancer Study Group and the Eastern Cooperative Oncology Group in the United States and the European Organization for Research on the Treatment of Cancer in Europe is conducting a prospective randomized trial comparing chemotherapy, operation, and radiotherapy with chemotherapy and radiotherapy. Until this trial is completed I believe operation should continue to be considered experimental for patients with stage IIIa SCLC.

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Operation for Small Cell Lung Cancer—Where Are We?

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The role of operation for small cell lung cancer (SCLC) has been hotly contested over the past 20 years. Despite misgivings promoted by the failure of operation to control disease as documented by a report of the British Medical Research Council in 1969 [1], surgeons continued to operate on SCLC. It soon became apparent that in stage I [T1 N0, T2 N0] disease, surgical therapy alone can provide curative treatment in up to 50% of patients. With the addition of postoperative chemotherapy, it appears that in this early stage of disease, even better long-term survival may be possible [2].

Even in more advanced disease (stage II and IIIA) when the tumor has spread to regional lymph nodes, the combination of complete surgical excision and postoperative chemotherapy and radiotherapy can afford 5-year survivals in 20% to 40% of patients [3]. Salzer and colleagues [4] report in this issue the results of 11 patients in whom N2 disease was identified preoperatively, who were then treated with preoperative chemotherapy, operation, and postoperative chemotherapy and radiotherapy, including prophylactic cranial irradiation. Three more patients also underwent resection and were treated postoperatively with similar intensive chemotherapy and radiotherapy. The projected 5-year survival rate for this small group of patients is 47%, compared with zero for a similar group of patients treated with operation and a variety of less aggressive postoperative therapies, including no further treatment. This neoadjuvant approach has recently been used by many groups. Unfortunately, no center has a sufficient sample of patients to afford meaningful results. Estimated 5-year survival rates range from zero to the most optimistic, as reported by Salzer and associates in this issue.

See also pages 691 and 759.

Does operation improve the results of treatment in early SCLC? Conventional treatment with chemotherapy, mediastinal irradiation and prophylactic cranial irradiation can be curative for "limited" SCLC. In recent reports, up to 20% of such patients treated conventionally are disease-free at 5 years. One cannot, however, compare these results with those of operation (which appear somewhat more optimistic), because "limited" SCLC includes many patients with more advanced (stage IIIA and IIIB) disease, for whom operation would never be considered part of the treatment program. In an attempt to identify the results of nonsurgical treatment in potentially operable patients, the Toronto group [5] demonstrated retrospectively that in "very limited" (stage I or II) disease, the 5-year survival rate of conventional therapy approaches 25%. For this analysis, all peripheral tumors were excluded. Additionally, this retrospective group of patients did not receive the most effective chemotherapy regimens used today. More information is needed about the results of conventional therapy in "very limited" resectible SCLC.

What then is the role of operation? Throughout the surgical literature it has been emphasized that in SCLC, surgical resection appears to reduce the local recurrence rate. With nonsurgical treatment, up to 30% of recurrences will be seen initially in the primary site, indicating the failure of chemotherapy and radiotherapy to fully control this site. In most surgical reports and in the present study by Salzer and associates, recurrence at the primary site as the first evidence of recurrence of disease appears to be in the range of 10%. Therefore, in early-stage SCLC, surgical resection may improve control of the primary tumor if a complete resection can be performed. The neoadjuvant treatment described in the report by Salzer and associates may downstage the tumor, thus increasing complete resectability. Whether or not radiotherapy after resection has a further advantage for local control is unknown.

Despite the optimistic report by Salzer and colleagues in this issue, there is no convincing evidence that surgical treatment is of added value in patients preoperatively identified as having SCLC with N2 involvement. The Lung Cancer Study Group has recently completed a randomized trial comparing conventional nonsurgical treatment with combined-modality neoadjuvant therapy, including operation, for limited SCLC. The results of this trial may help to shed light on the dilemma we face at the moment.

Should operation be recommended in limited SCLC? Certainly clinical stage I SCLC patients should be offered surgical resection, and if the histology is confirmed at operation, postoperative chemotherapy and radiotherapy is recommended, especially if, as frequently happens, the ultimate pathological stage is II or IIIA. Every attempt should be made during the surgical procedure to effect a complete resection. However, when more locally advanced (stage II and III) SCLC is identified preoperatively, there is no evidence as yet to recommend the addition of operation to conventional therapy, unless the histology is

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in question. The role of neoadjuvant chemotherapy in this stage of disease is still speculative. This approach should still be reserved for clinical trial settings.

One must keep in mind that of all patients seen with limited SCLC, only about 10% will have sufficiently early disease to warrant consideration for surgical treatment.

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HONORED GUEST ADDRESS

Research and the Practicing Surgeon

Mark V. Braimbridge, FRCS

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Introduction of Mr Braimbridge by President Richard E. Clark

One of the real delights of being President is to have the opportunity to have an invited speaker. All of us have a great number of friends, I am sure, and when one begins to think about this a little bit, one always gets, I guess, into a little bit of an indecisive situation. My decision was easy. Let me tell you a little bit about this man who is going to talk to us.

He is the son of a Nairobi surgeon and spent his early years growing up in Africa. He was educated at Cambridge and had his medical training at St. Bartholomew's. He had an opportunity to work with Frank Gerbode at Stanford in San Francisco, when Stanford was still there, and has been an erstwhile surgeon and working at St. Thomas' through all these years.

He served on many European editorial boards. He was a founding member of an unusual club that is called Pete's Club. It is a European traveling club of surgeons where you only present a case, it is a case meeting, but the presentation should not reflect credit on the presenter. He has also been President of the only society of cardiothoracic surgery in Great Britain and Ireland 3 years ago, in 1986.

Why Mark Braimbridge? I will tell you why. He is a man of incredible inquiry, a deep, broad education. He speaks multiple dialects of Swahili,[*] *Greek, and French; not only is he a linguist, but he has an enormous sense of history of Europe. And he is always a man who I found constantly was striving to learn from others all over the world and to bring these new ideas and techniques to his patients. He has been in his own right a remarkable innovator. I have had the wonderful pleasure of seeing him operate in two hospitals. Mark is absolutely unflappable. He is extremely orderly, and he has truly bridged the gap from the craftsman surgeon to the artisan surgeon. So I give you a world-class cardiothoracic surgeon, a great teacher, and my friend, Mark V. Braimbridge.*

The subject of research and the practicing surgeon has been particularly relevant to my practice over the last 30 years. It is also topical because Francis Robicsek this year has stated that "the United States has lost its pre-eminence in cardiothoracic research" (personal communication, Munich, Sep 1989). If this is true, it makes the article by Professor Michael Oliver from Edinburgh on "Crisis in Cardiovascular Research in Britain" [1], which appeared in the *British Heart Journal* this year, particularly ominous.

Factors Seducing Young Surgeons From Research

St. Thomas' Hospital in London was founded by the Benedictine monks in 1187. This may account for the somewhat traditional attitude of one of the consultant surgeons, who would never appoint a resident if he had a beard, wore a bow tie, or smoked a pipe, as he felt that these indicated an unfortunate propensity toward research.

This attitude is not only traditional. This year a surgeon at Guy's Hospital in London said to me, "Research is for researchers; my registrars train to become clinical National Health Service surgeons."

Research for Curriculum Vitae Only

This unhelpful attitude is one of the many reasons for the initial "bimbo factor" seducing young men away from a career in research. They are told that they should do research only to fill in the research section of their curriculum vitae and then should forget about it.

Reduced Government Funding

The second seductive factor is that government research funds in Britain, and I gather in the United States also, have been markedly reduced.

Clinical Workload

The third important factor is the young man saying, "I have too much of a clinical workload." This factor, however, is put into perspective by a famous American[†] who said, "Men will go to almost any lengths to avoid having to think." There is no question but that operating is one of those lengths.

Higher Salaries of National Health Service/Prepaid Plans

Even more seductive in keeping young men away from research are the higher salaries in Britain of the National Health Service, and, I understand, of the prepaid plans in the United States.

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[*] Swahili, *not* Arabic.

[†] I cannot locate this reference in either Emerson or Voltaire, both of whom have been quoted as its author.

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Fig 1. Dr Frank Gerbode.

Fig 2. Professorial transport, Cambridge University.

Private Practice

Most of all, the lure of private practice and its attendant income is the strongest factor.

Necessary Qualifications for a Consultant Appointment in the National Health Service in the 1960s

The qualifications I had to have as a young man to become a consultant in the National Health Service in Britain in the 1960s was MA, MB, BChir, which meant that I had qualified as a doctor at Cambridge and St. Bartholomew's Hospital; FRCS(Eng) meant I had passed my English general surgical boards; and I also had to have at that time my BTA ("Been to America"). I was very fortunate in 1956 in coming to Stanford in San Francisco, where I underwent a fundamental conversion toward research owing to the influence of that great man, Dr Frank Gerbode (Fig 1).

I find now, as I did then, that surgeons who are not in the university service say, when I speak to them, "University surgeons are paid to do the research, not me: and when I am confronted with the Nobel Prize/Trump Tower dilemma, there really is no contest." This was brought home to me earlier in my career by the fact that the mode of transport of my professor at Cambridge was his wife's bicycle (Fig 2), whereas a 1990 American specification Rolls Royce Corniche III (Fig 3) is, I understand, the town runabout of most of the members of this Society.

Medical Marriage Syndrome

There is another problem—the medical wife. There are three stages of the medical marriage syndrome as described in the literature. Characteristically, the woman who marries a doctor is a type who seeks attention, and she falls for the private patient "Darling, lie down on the couch and tell me about yourself" type of treatment. They then marry, and it becomes, "Darling, you look after the home and I will go out and earn the money," and he disappears into the operating room. Ten years later, stage three begins and what the literature calls a "wild, wild woman" is in constant conflict with an increasingly withdrawn surgeon. In fact, one of my colleagues used to drive home, park his car in the garage, and sit there for two hours before he could pluck up the courage to go indoors!

Career Choice

So when I was faced, aware of these factors, with being a university surgeon with time but no money or having private practice with money but no time, I opted for the second when a National Health Service consultant appointment came up at St. Thomas' Hospital in London. The influence of Frank Gerbode was, however, such that I felt it essential, if I could, to continue in a research capacity of some sort.

Surgical Research Options

One may divide surgical research into three categories: the "adding up numbers" research—retrospective clinical studies; prospective clinical studies; and laboratory research.

Fig 3. 1990 American specification Rolls-Royce Corniche III.

Retrospective Studies

Many young surgeons, when asked about "numbers" research, tend to say, "I have too few cases; I don't have a statistician to help me; in any case, I have no time to write up and present my data; and I have no computer service, in the sense that I have no one to input the data or take it out."

Prospective Clinical Studies

When asked about prospective clinical studies, they say, "I have no one to take the samples," though I have found that it is surprising how much can be done by a friendly nurse. They say, "There is no money for processing samples, either." But one can "bribe" a pathologist by putting his or her name on the paper, and even private hospital authorities will fall for the lure of, "If you put this in the educational section of your annual report, it will show your Board how much you support research."

There is no doubt, however, that prospective studies are a litigation minefield in the United States. Shortly before travelling to Phoenix, I received, from a colleague of Hendrick Barner, the Investigation Review Board form for a prospective clinical study in St. Louis. It would have taken me a fortnight just to fill in the form before even starting the project. It is a different format altogether in the United States compared with Europe, because in the United Kingdom all that is required is to write a letter to the hospital ethical committee. Our patients also still believe that doctors know best, making obtaining consent simple. Most of all, our lawyers cannot accept a contingency fee.

Laboratory Research

Laboratory research is immediately, as a rule, written off by nonuniversity surgeons, but I would like to suggest that this is the way for us to go. In order to be confident of getting a paper in the peer-reviewed literature these days, every author—that is, apart from the freeloaders—must be a recognized authority. Now surgeons are only authorities on surgery. If we surgeons want to get our fundamental questions answered, we have to go to the basic scientists to get them to do our laboratory research for us, because we ourselves have neither the money nor the time. Such research is necessarily multidisciplinary.

Components of Successful Multidisciplinary Research

After consultation, the basic scientists with whom I have worked over the years and I have identified five ground rules for successful, multidisciplinary laboratory research by the practicing, nonuniversity surgeon.

Communication

The first important factor is communication. The surgeon must be accessible, which in practice means he should visit the laboratory once a week, say, and talk to everybody. In addition, the basic scientist should see the stress under which the surgeon operates and should come into the operating room himself at times.

Mutual Respect

The second factor is that there should be mutual respect. Surgeons treat basic scientists, they feel—and I think rightly—as tame technicians. The standard quotation they hear is some such as, "I'll go hire myself a biochemist." But the basic scientists also, I have to point out to them, treat the surgeon as educationally subnormal, with the sort of comment, "Even a surgeon should understand that!"

Support for the Basic Scientist

The third factor is that the surgeon should support his basic scientist, because he may be in limbo between basic science and clinical practice.

POLITICAL SUPPORT. He should support him politically; that is, write to his dean about his career structure and support him in this in every way he can.

FINANCIAL SUPPORT. He should support the basic scientist financially, by which I mean he should support his fund-raising efforts and allow him to apply with joint applications to sources of surgical research funds.

LITERARY SUPPORT. There should be mutual literary support to stop a surgeon making stupid statements in a basic scientific journal or the basic scientist making idiotic remarks in a surgical journal.

Clarity of Presentation of the Surgical Problem

The most important factor, we felt, was the clarity of presentation of the surgical problem to the basic scientist in order to obtain an equally clear answer. The surgeon has to realize that any research is stimulating to a basic scientist. The hearing mechanism of a snail is intrinsically just as exciting as myocardial biochemistry. But there is no question that the clinical application of basic science is a major bonus to that scientist himself. I would like to give some examples from our experience of the importance of clarity of presentation of surgical problems.

"WHY ARE OUR PATIENTS DYING OF LOW CARDIAC OUTPUT?" In 1960, when I was working as senior registrar to Sir Russell Brock at the Brompton Hospital, I went to the Royal College of Surgeons, Lincoln's Inn Fields, London, and I met there two cytochemists, Drs Joe Chayen and Lucille Bitensky (Fig 4). I asked them, "Why are our patients dying of low cardiac output with normal myocardial histology?" They said, "Send us some biopsy specimens of the left ventricle frozen to −70°C in hexane [2]: We'll look at frozen sections with our polarizing microscope, because we have found that in air, myosin is disoriented, polarized light does not come through, and, in normal muscle, the section looks dark. When we add adenosine triphosphate (ATP) and calcium, the myosin becomes oriented, the polarized light is allowed through, and the section immediately becomes brighter. Damaged or ischemic muscle, on the other hand, is already much brighter and more oriented in air, so that adding ATP and calcium makes less difference. This difference is smaller than that in the undamaged muscle" [3, 4].

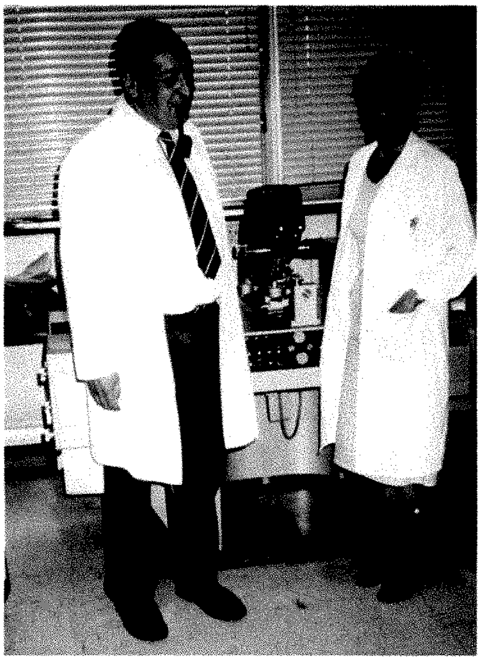

Fig 4. Drs Joe Chayen and Lucille Bitensky.

To validate this quantitative birefringence test, we asked our cardiologists to separate the left ventricular function of a series of aortic valve patients at the time of cardiac catheterization into normal and abnormal myocardium by eight physiological tests: the left ventricular end-diastolic pressure and volume, ejection fraction and rate, cardiac index, peak velocity of the contractile fraction, KV_{max}, and KV_{max} on a postectopic beat [5]. We asked them also to take transseptal endomyocardial biopsies of the left ventricle at the same time. We found we were able to separate these two groups of good and poor physiological left ventricular function by the increase in birefringence in response to ATP and calcium with a probability of 0.001.

Applying this test to a series of aortic valve replacements carried out over a 24-year period, we found that the cellular chemical assessment of preservation during bypass correlated closely with clinical outcome in terms of low cardiac output and death [6]. This relationship held good whether we were using intermittent perfusion with 15°C blood or 30°C blood in the 1960s or when we changed to continuous coronary perfusion, first at 30°C then at 32°C with a beating heart, or with single-infusion cardioplegia or, finally, multiple-infusion cardioplegia. It is only now, with multiple-infusion cardioplegia, that we have satisfactory biopsy and clinical outcomes in these patients.

The simple answer to our question, therefore, about the cause of cardiac surgical mortality came from Dr Darracott-Cankovic, who had been put onto working with us full time in 1962. It was, "You're damaging the myosin."

"HOW DO I PROTECT THE MYOSIN?" In 1973 I met Sir Ernst Chain, the biochemist of the penicillin trio who received the Nobel prize for their work (Fig 5). I asked him, "How do I protect the myosin that I've found that I am damaging, and, if you can tell me, what will it cost me to find out?" His answers were, "Ask a Research Fellow working in my laboratory, Dr David Hearse," and "It will cost you nothing." David Hearse (Fig 6) reminds me that my exact words to him were, "Is potassium good for the heart?" I had asked him this because of the work of Bretschneider in Germany, of which we had heard [7].

Fig 5. Sir Ernst Chain, Nobel laureate. (Reprinted by permission from Clark RW. The Life of Ernst Chain. London: George Weidenfeld & Nicholson, Ltd.)

Dr Hearse took this problem to his isolated working rat heart preparation, his first publications appearing in 1974 [8]. Standard protocol was to establish control aortic flow rates, infuse the cardioplegia being investigated followed by the ischemic interval, and then reperfuse the heart and express recovery as a percentage of control [9].

Using this technique, he found that if he infused buffer for those two minutes, there was no recovery after 30 minutes of normothermic ischemia. If he added 16 mmol/L of potassium there was a 29% recovery; adding ATP, 77%; 16 mmol/L of magnesium, 87%; adding creatine phosphate and procaine, 93%; and if he then reduced the

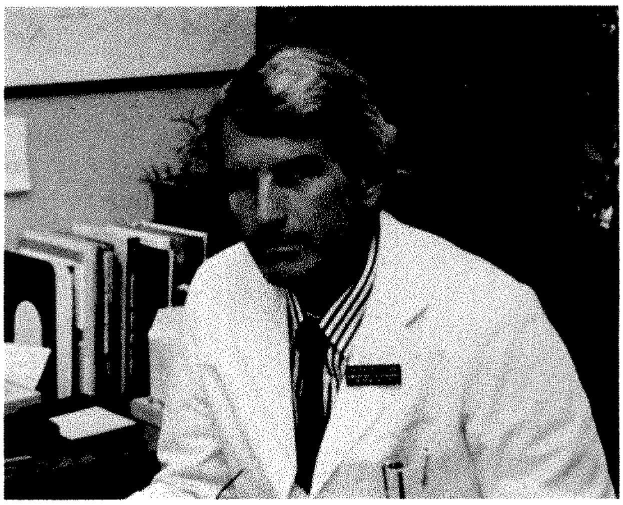

Fig 6. Professor David Hearse, Rayne Institute, St. Thomas' Hospital, London, England.

temperature to 30°C, he had a 98% recovery. But he pointed out that this does not mean one can just add, say, magnesium period, because, being a biochemist, he had done dose-response curves for every component that he investigated, and found that they were roughly bell-shaped curves. The optimum magnesium concentration, for instance, in the cardioplegia was 16 mmol/L [10, 11], protection falling off markedly on each side of this concentration.

So his answer to me shortly after I had asked him the question, "Is potassium good for the heart?" was "Potassium is good [7], magnesium is good [12], glucose and insulin are not [13], and calcium antagonists work at normal temperatures, but are of no advantage at 20°C [14–16]. But," he added, "do remember the dose-response curve [12, 13] and don't add odd components at odd concentrations for theoretical reasons [17]."

So we evolved the clinical St. Thomas' solution in 1975 at the start of our clinical cardioplegic program, adding to a liter of 4°C Ringer's solution an ampoule that contained 16 mmol/L of potassium chloride, making it up to 20 mmol/L, 16 mmol/L of magnesium chloride, and one millimole of procaine.

"WHICH MYOSIN HAVE I BEEN DAMAGING? I then went back to the cytochemists and said, "If I am damaging the myosin, tell me which myosin I am damaging." So we divided the left ventricular biopsy specimens into outer and inner halves. Dr Darracott-Cankovic showed that when I was using continuous coronary perfusion at 32°C, I was only preserving 25% of both the inner and outer halves of the myocardium. In 6% I was damaging both, but, most importantly, 63% of the biopsy specimens showed damage of the endomyocardial half of the specimen. When we changed to single-dose cardioplegia, we had better protection of both halves of the myocardium and had abolished the endomyocardial deterioration. However, we now had more deterioration of the outer half of the myocardium. By changing to multidose cardioplegia and concentrating on topical pericardial cooling, we were able to get a satisfactory outcome from both halves of the biopsy specimen [18]. I was therefore told, "The myosin you are damaging is the endomyocardium, unless you use multidose cardioplegia and topical cooling."

"SHOULD I OXYGENATE THE ST. THOMAS' CARDIOPLEGIC SOLUTION?" I then went back to Professor David Hearse in 1986, because there was a lot of talk about the advantages of oxygenation of crystalloid cardioplegia, to ask, "Should I oxygenate my cardioplegic solution?" He looked at our original 1975 clinical solution in the rat heart after three hours of hypothermic arrest. We found that, with this solution, there was only 20% recovery. If we oxygenated the solution, there was 62% recovery [19].

By this time Abbott Laboratories were making the St. Thomas' solution commercially under the name of Plegisol, using rather less sodium chloride, a little less potassium chloride (16 instead of 20 mmol/L), 16 mmol/L (the same) of magnesium chloride, and half the calcium (1.2 mmol/L) [20, 21]. This time the solution was buffered with sodium bicarbonate to pH 7.8.

If we used Plegisol without oxygen, there was a markedly better recovery than that of our initial solutions even with oxygen. In order to check whether oxygenation was important for Plegisol, we had to increase the ischemic time to four hours, when we were able to show some improvement by oxygenating Plegisol.

So, the answer to my question, "Did I need to oxygenate my cardioplegic solution?" was, "Yes, unless you use Plegisol, and even then, only if you anticipate going to four hours of ischemia." We have recently completed a prospective clinical study that shows that, in fact, on clinical assessment there is little advantage in oxygenating Plegisol, although there is a slight improvement in biopsy assessment.

"DOES THIS APPLY TO CARDIAC TRANSPLANTATION?" Mr Terence English, who initiated at Cambridge the cardiac transplantation program in the modern era in England, knew of the work of Dr Darracott-Cankovic with biopsy assessment of the St. Thomas' solution. He asked, "Did it apply to cardiac transplantation?"

Dr Darracott-Cankovic was supplied with biopsy specimens from 172 donor hearts: from the donor before the heart was harvested, after transport to the recipient, and at the end of bypass. She found that 73 of the donor hearts already had damaged myocardium before harvesting from the donor, and, of these, 51% had a low cardiac output postoperatively; at the time of study, 45% of these patients had died. She found that 47 of the 172 patients had deteriorated on biopsy during transport, having been good when they started. Fifty-one percent of these also had low cardiac output postoperatively, and 40% had died. If, however, the donor heart biopsy was satisfactory on harvesting and the heart had not deteriorated during transport—there were 52 of these—only 4% suffered from a low cardiac output and only 6% died [22].

Her answer to Terence English, therefore, as to whether our work on the St. Thomas' solution applied to cardiac transplantation also was, "No, you'll have to think again." He is now therefore investigating pretreating the donors with the Cape Town thyroxine regimen before harvesting, and modifying the protective regimen for the donor hearts.

Attitude of Junior Staff

The last of the criteria for successful multidisciplinary research applies to junior staff. If they are involved, they should be given a precise protocol, and, particularly, they should be educated in the attitude to the basic scientist that the senior scientist already has.

A surgical registrar, working in the Rayne Institute recently, tended to behave, when frustrated, like a normal cardiac surgeon under stress—shouting, swearing, breaking glassware—which went down extremely badly with the Institute Cardiovascular Research staff (Fig 7), who quickly told him to behave like a basic scientist, or go!

Fig 7. Cardiovascular research, Rayne Institute, St. Thomas' Hospital, London, England.

Necessity for Surgical Drive

It is nevertheless necessary to have a degree of drive if a practicing surgeon is going to push through such projects. One can, in this context, separate drive into three components. Almost every surgeon has the ability to initiate a project. Some find it difficult to develop the ability to maintain momentum when the going gets rough. It is, however, the third component that most often stops the surgeon from finishing his project: the ability to make his colleagues like it. It is mainly because of this third component of surgical drive that we have outlined the above five ground rules that we feel are so important for successful multidisciplinary research.

Sacrifices of a Practicing Surgeon

Finally, the practicing surgeon must make sacrifices if he is going to embark on multidisciplinary laboratory research.

Time

He has to sacrifice time. I rarely, however, find it necessary, once the project is underway, to spend more time than two hours per week in the laboratory. Usually it is less.

Money

Multidisciplinary research will cost $1,000 per year for stamps, telephone calls, and dinners for arriving and departing foreign Fellows who have been attached to surgical projects.

Coffee Consumption

I have, however, found it necessary to consume liters of coffee in sorting out the angles of some of these projects. It is one thing being a project's head; it is quite another being its stomach.

Complacency

One thing I have had to give up completely, however, is complacency. I have never been able to say, as a lot of young surgeons, I note, are saying, "I know how to do this operation; please don't fuss me with fancy theories."

Conclusion

In conclusion, if we are going to avoid this quotation from that same *British Heart Journal* article [1], "There is a dearth of clinical scientists both in the United Kingdom and in North America," I believe strongly that it is up to us, the nonuniversity surgeons, to help to fill this gap. May I suggest that the next time a young man with ambition comes to you for advice when faced with the dilemma of whether he should aim for a Nobel prize or the Trump Tower, he should be encouraged to go for both!

I would like to thank the Association once again for the great privilege you have allowed me of being able to present to you this small token of esteem for that great American surgeon, Dr Frank Gerbode.

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Heart Valve Operations in Patients With Active Infective Endocarditis

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Sixty-two consecutive patients underwent heart valve operation for active infective endocarditis. There were 42 men and 20 women whose mean age was 49 years (range, 21 to 79 years). The infection was in the aortic valve in 37 patients, the mitral valve in 18, the aortic and mitral valves in 5, and the tricuspid valve in 2. Twenty-four patients had prosthetic valve endocarditis. *Staphylococcus* and *Streptococcus* were responsible for 86% of the infections. Annular abscess was encountered in 33 patients. Complex valve procedures involving reconstruction of the left ventricular inflow or outflow tract or both were performed in 31 patients. There were three operative deaths (4.8%). Predictors of operative mortality were prosthetic valve endocarditis, preoperative shock, and annular abscess. Patients were followed for 1 month to 130 months (mean follow-up, 43 months). Only 1 patient required reoperation for persistent infection. There were ten late deaths. Most survivors (96%) are currently in New York Heart Association class I or II. The 5-year actuarial survival was 79% ± 7%. These data demonstrate excellent results in patients with native valve endocarditis, and support the premise that patients with prosthetic valve endocarditis should have early surgical intervention.

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Infective endocarditis of the heart valves is often cured with antibiotics [1, 2]. The characteristics of the offending microorganism and the location of the heart valve in which it is nested are probably the two principal determinants of the outcome of antibiotic therapy. Certain microbes do not respond to antibiotics alone, and the same microorganism can react differently depending on the site of the valve in which it is lodged. For instance, endocarditis caused by *Staphylococcus aureus* can usually be cured with antibiotics alone when the native tricuspid valve is involved, but surgical intervention is frequently necessary when the native aortic valve is the site of infection. Antibiotics can be effective in eliminating a microorganism from a native valve but ineffective against the same microorganism in a prosthetic heart valve.

Thus, although antibiotics are the principal treatment of infective endocarditis, operative intervention is an indispensable adjunct in many patients during the active phase of the infection. The difficulty is to determine when to intervene. As the results of valve surgery improve, a more aggressive approach to the management of these patients seems reasonable. This article summarizes our experience with patients who required a heart valve operation during the active phase of infective endocarditis.

Material and Methods

A review of our valve surgery registry indicated that from January 1978 to June 1989, 62 patients underwent heart valve operation during the active phase of infective endocarditis. Every patient was operated on during the course of antibiotic therapy or even before the diagnosis of infective endocarditis had been established. There were 42 men and 20 women whose mean age was 49 years (range, 21 to 79 years). The principal indications for operation are shown in Table 1. Emergent operation was necessary in 12 patients in septic or cardiogenic shock or both. In addition, symptoms of congestive heart failure were present in 45 patients (73%), sepsis in 34 (55%), peripheral emboli in 25 (40%), cerebral emboli in 8 (13%), and myocardial infarction in 3 (5%). The heart failure was severe in 26 patients and moderate in 19.

The preoperative electrocardiogram revealed sinus rhythm in 47 patients, atrial fibrillation in 12, and complete heart block in 3. Five patients had conduction disturbances (3 with increased PR interval and 2 with complete heart block) suggestive of annular abscess. Three other patients had evidence of recent myocardial infarction.

Thirty-eight patients (61%) had native valve endocarditis. Eleven of them had preexisting valvular lesions: aortic valve disease in 5 (bicuspid in 3 and rheumatic in 2), mitral valve disease in 4 (myxomatous in 3 and rheumatic in 1), and aortic and mitral valve disease in 2 (rheumatic). Antecedent events thought to be responsible for the endocarditis were identified in 14 patients: dental procedures in 8, remote infection in 3, and drug abuse in 3.

Twenty-four patients (39%) had prosthetic valve endocarditis; 8 had mechanical valves and 16, tissue valves. Four patients had had composite replacement of the ascending aorta and aortic valve. The endocarditis occurred early (within 6 months from the original operation)

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Table 1. Indications for Operation, Incidence of Annular Abscess, Microorganisms, and Operative Survival

	Native Valves				Prosthetic Valves			
Variable	Aortic	Mitral	Aortic + Mitral	Tricuspid	Aortic	Mitral	Aortic + Mitral	Total[a]
No. of patients	21	13	2	2	16	5	3	62 (100)
Indications for operation								
Shock	3	2	0	0	6	1	1	13 (21)
Heart failure	11	6	1	0	4	2	1	25 (40)
Sepsis	6	4	1	2	5	2	1	21 (34)
Emboli	1	1	0	0	1	0	0	3 (5)
Annular abscess	12	3	0	0	13	3	2	33 (53)
Microorganisms								
Staphylococcus aureus	10	5	0	0	1	2	0	18 (29)
Staphylococcus epidermidis	0	1	0	0	2	3	2	8 (13)
Streptococcus viridans	6	5	1	1	2	0	0	15 (24)
Streptococcus faecalis	3	0	0	1	1	0	1	6 (10)
Other streptococci	2	1	0	0	3	0	0	6 (10)
Pseudomonas	0	0	0	0	2	0	0	2 (3)
Other bacteria	0	0	0	0	5	0	0	5 (8)
Undetermined	0	1	1	0	0	0	0	2 (3)
Operative survival	21	13	2	2	15	4	2	59 (95)

[a] Numbers in parentheses are percentages.

in 9 patients and late in 15. Antecedent events were identified in 8 of the 15 patients with late endocarditis: dental procedures in 5, drug abuse in 2, and remote infection in 1. Seventeen patients had had valve replacement once, 5 twice, and 2 three times before the operation for infective endocarditis.

Blood cultures were positive in 58 patients and negative in 4. All surgical specimens were sent for culture, and microorganisms were isolated for 39 patients, including 2 of the 4 patients in whom blood cultures were negative. Thus, the microorganism responsible for the infection was identified in 60 patients. Table 1 shows the bacteria that caused endocarditis in these patients. Eight patients had received antibiotics for less than one day; the remaining patients had been on a regimen of antibiotics for one day to 27 days (mean duration, 11 days) when operation was performed.

Echocardiography was performed in all patients before operation. It revealed vegetations in the leaflets of 29 patients with native valve endocarditis and vegetations or abnormal masses in the leaflets of 6 with a bioprosthesis. In addition, the diagnosis of aortic root abscess was made by echocardiography in 8 patients; 5 had a fistula between the aortic root and surrounding structures, and 3 had a paravalvular cavity suggestive of a false aneurysm.

Eight patients underwent heart catheterization and coronary angiography before operation. An additional 9 patients with early prosthetic valve endocarditis had had coronary angiography before the original heart valve procedure. Major coronary artery disease was present in 6 of these 17 patients.

Operations

The operations performed are summarized in Table 2.

Patients with infection limited to the leaflets of the native or bioprosthetic valve were treated by valve replacement except for a few with tricuspid or mitral valve endocarditis in whom valve reconstruction was feasible. If the infection had extended into or beyond the annulus, aggressive debridement of all infected and inflamed tissues and reconstruction of the left ventricular inflow and outflow tracts and the atrial wall with either autologous or glutaraldehyde-preserved bovine pericardium was performed.

When the abscess was limited to the aortic annulus, simple excision of that portion of the annulus and corresponding sinus was done. Reconstruction of the aortic root was performed with a properly tailored patch of pericardium secured to the healthy tissue around the defect by a continuous, running 4-0 polypropylene suture.

When the abscess extended through the aortic wall into other tissues or cavities, a more extensive resection was performed. If the interventricular septum was involved, no serious consideration was given to conduction pathways, as the primary concern was to extirpate all necrotic and potentially infected tissues around the abscess cavity. In cases in which the abscess was in the ventricular muscle, the patch was secured to the left ventricular endocardium with a running 4-0 polypropylene suture. An aortic valve prosthesis was then secured to the aortic annulus in unresected areas and to the pericardial patch in areas where the annulus had been reconstructed.

Table 2. Operations Performed

Operations	No. of Patients^{a}
Aortic valve endocarditis	37 (100)
AVR	12^{b} (32)
RLVOT + AVR	12^{b} (32)
RLVOT + composite AVR and RAA	5 (14)
RLVOT + AVR + RAA	2 (5)
RLVOT + AVR + RAA + MVR	2 (5)
RLVOT + composite AVR and RAA + MV repair	1 (3)
RLVOT + composite AVR and RAA + MVR	1 (3)
RLVOT + AVR + MV repair	1 (3)
RLVOT + AVR + RRVOT + PVR + TVR	1 (3)
Mitral valve endocarditis	18 (100)
MVR	13^{c} (72)
MVR + RMA	4 (22)
MVR + AVR	1 (6)
Aortic + mitral valve endocarditis	5 (100)
AVR + MVR	3 (60)
RLVOT + RMA + MVR + AVR	2^{c} (40)
Tricuspid valve endocarditis	2 (100)
TV repair	2 (100)

[a] Numbers in parentheses are percentages. [b] Two patients also had aortocoronary bypass. [c] One patient also had aortocoronary bypass.

AVR = aortic valve replacement; MV = mitral valve; MVR = mitral valve replacement; PVR = pulmonary valve replacement; RAA = replacement of ascending aorta; RLVOT = reconstruction of left ventricular outflow tract; RMA = reconstruction of mitral annulus; RRVOT = reconstruction of right ventricular outflow tract; TV = tricuspid valve; TVR = tricuspid valve replacement.

In a number of patients, the aortic root abscess extended into the fibrous skeleton that anchors the base of the anterior leaflet of the mitral valve, and in some patients, the inflammation had destroyed most of the anterior leaflet, thus making mitral valve replacement also necessary. In these patients, after the abscess was excised, the aortic and mitral annuli became a single orifice, and reconstruction was done with a triangular pericardial patch [3, 4].

Patients with multiple abscesses in the aortic annulus ended up with complete aortoventricular separation after debridement, and reconstruction of the left ventricular outflow tract was accomplished with a circumferential pericardial patch 1 to 3 cm high. One or both coronary arteries had to be reimplanted into this patch in several patients. Others required replacement of the proximal ascending aorta; this was done by suturing a composite graft with an aortic prosthesis directly to the pericardial patch [5]. Twelve of 21 patients with native aortic valve endocarditis and 13 of 16 patients with prosthetic aortic valve endocarditis were found to have annular abscess at operation (see Table 1).

Patients with an abscess of the mitral annulus represented a greater challenge, and until 1984, we simply debrided the abscess and secured the prosthetic valve in the surrounding atrial or ventricular muscle. We saw 2 such patients, and the results were not satisfactory in either: 1 died soon after operation, and in the other a recurrent infection developed; after reoperation, he was left with a leaking prosthetic mitral valve. A technique of mitral annulus reconstruction was then developed, and the results have been most gratifying [3, 4].

With this technique, the mitral annulus abscess is aggressively debrided, and the defect created is corrected with a properly tailored autologous or bovine pericardial patch [3]. The simplest situation is when a small segment of the posterior part of the mitral annulus is destroyed by infection. After appropriate debridement, a semicircular patch of pericardium measuring 1 cm longer and 1 cm wider than the defect in the annulus is prepared. The curved side of this patch is sutured to the endocardium of the left ventricle around the abscess cavity, starting and finishing at the normal fibrous annulus. We use a running 4-0 polypropylene suture, and the bites to the endocardium should be deeper in the trabeculated areas but never transmural, as they may damage major coronary arteries. The mitral valve prosthesis is secured to the healthy mitral annulus and to the straight side of the patch.

More extensive destruction of the mitral annulus by multiple abscesses is better treated by circumferential reconstruction of the mitral annulus. A strip of pericardium is sutured to the endocardium of the left ventricle posteriorly and to fibrous skeleton superiorly. The mitral prosthesis is secured entirely in the pericardial patch [3, 4].

Simultaneous reconstructions of the aortic and mitral annuli are possible with a single patch sutured to the base of the left ventricle [3–5].

Statistical Analyses

Univariate analysis, with operative mortality as the end point, was performed on clinical, echocardiographic, bacteriological, and operative variables. A stepwise logistic regression analysis was applied to significant variables.

Results

There were three operative deaths, an overall operative mortality of 4.8%. All three deaths occurred in patients who had prosthetic valve endocarditis and were in shock when they underwent operation. Two deaths were due to low cardiac output syndrome after a prolonged operation in patients who required aortic and mitral valve replacement for multiple abscesses. The third death was due to hepatic failure in a cirrhotic patient who required aortic, tricuspid, and pulmonary valve replacement for multiple abscesses. Thus, the operative mortality for patients with prosthetic valve endocarditis was 12.5%; there were no operative deaths among patients with native valve endocarditis. By univariate analysis, the following variables were predictive of operative mortality: prosthetic valve endocarditis, preoperative shock, and annular abscess. Stepwise logistic regression analysis indicated that only prosthetic valve endocarditis was significant.

Postoperative complications were common. Nine patients required reexploration of the mediastinum for excessive bleeding or tamponade; no bleeding source was found in 8 patients, and it was attributed to coagulopathy.

Five patients required assisted ventilation for more than two days. Two patients sustained a perioperative stroke; 1 recovered completely and 1, only partially. Low-output syndrome occurred in 5 patients. Seven patients required dialysis for renal failure. Nine patients required a permanent transvenous pacemaker because of heart block. Two patients underwent amputation of a leg for control of sepsis, 1 below and 1 above the knee. Three patients had electrocardiographic evidence of myocardial infarction, but it was present before operation.

All patients received a total of at least 4 weeks of intravenous antibiotics. Patients in whom the surgical specimen was sterile received antibiotics for at least 1 week after operation but no less than a total of 4 weeks of preoperative and postoperative treatment. When the surgical specimen grew bacteria, antibiotics were given for 4 postoperative weeks.

Patients who had mechanical valves implanted were placed on a regimen of Coumadin (crystalline warfarin sodium) permanently. Patients with porcine valves received anticoagulants during the first 3 postoperative months.

Forty-nine patients (79%) are alive after a mean follow-up of 43 ± 34 months (range, 1 to 130 months). Thirty-eight patients (78%) are functionally in New York Heart Association class I; 9 (18%) are in class II; and only 2 (4%) are in class III.

There have been ten late deaths, five of which were cardiac related (only 1 was valve related). Two patients were lost to follow-up at 12 and 36 months. One patient had recurrent mitral valve endocarditis because of mitral annulus abscess. He underwent reoperation, and was cured of his infection. However, perivalvular dehiscence developed, and he died in congestive heart failure 2 years later. This happened before techniques for mitral annulus reconstruction [3-5] were available. Two patients required valve rereplacement for structural valve deterioration of a porcine bioprosthesis (both mitral valves) after 8 years and 10 years, and are currently asymptomatic.

All patients have had a recent Doppler echocardiographic study. No patient has evidence of prosthetic valve dehiscence. One elderly woman has a mitral bioprosthesis with signs of degeneration, but she is not a candidate for reoperation.

Six patients have sustained postoperative strokes (2 with mechanical and 4 with tissue valves).

The actuarial survival is shown in Figure 1. The actuarial 5-year survival for all patients was 79% ± 7%; for patients with native valve endocarditis, it was 86% ± 8%, and for patients with prosthetic valve endocarditis, 67% ± 11% (p = not significant).

Comment

Appropriate antibiotic therapy can eradicate the infection in most patients with native valve endocarditis [1, 2]. Surgical intervention before completion of a standard course of antibiotics is reserved for patients in whom heart failure, persistent sepsis, recurrent emboli, annular abscess, or a combination of these develops [1]. The probability of operation being necessary during medical treatment is greater in patients with left-sided endocarditis than those with right-sided endocarditis. On the left side of the heart, the aortic valve requires surgical intervention more often than the mitral valve [1, 2]. Infections caused by certain microorganisms are also more likely to require surgical intervention because of their power to destroy tissue, eg, *S aureus*, or their resistance to antimicrobials, eg, *Serratia*, *Pseudomonas*, and *Candida* [1, 2, 6-8].

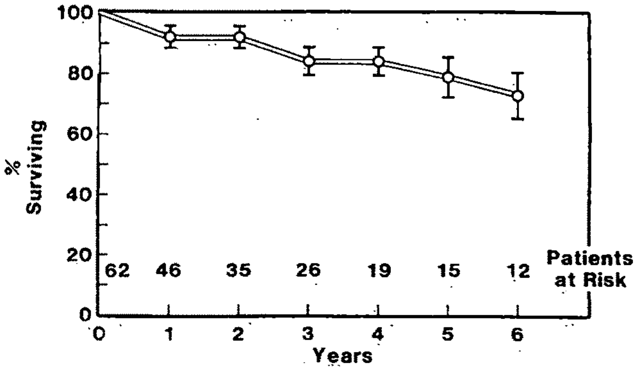

Fig 1. Actuarial survival of patients with active infective endocarditis.

There were no operative deaths among our patients with native valve endocarditis. These results are better than others previously reported [6, 7], probably because the operations were performed during the past decade when improved methods of myocardial protection and better techniques to treat patients with annular abscess were available [3-5]. In addition, all infections in our patients were caused by bacteria. The operative risk in patients infected with fungus is apparently high [1, 6, 7].

Prosthetic valve endocarditis has always been associated with greater mortality and morbidity than native valve endocarditis [6, 8]. Antibiotics alone seldom eradicate infection in prosthetic valves [6, 8-10]. The pathological process of prosthetic valve endocarditis is different from that of native valves. Infection in a mechanical valve is always located in its sewing ring [11]. In aortic valve homografts, the infection begins in the leaflets and destroys them very rapidly with consequent severe valvular dysfunction [12]. It seldom spreads into the annulus. Infections in porcine and pericardial bioprostheses behave differently depending on when they occur. Early infections involve the annulus or the leaflets or both. Late infections are frequently localized in the leaflets [10]. Núñez and associates reported on 20 patients who had bioprosthetic valve endocarditis [10]. Antibiotics alone cured 9 patients who did not have any echocardiographic evidence of valve dysfunction. Most of these infections were caused by streptococci. For the remaining patients, the valve was examined either at autopsy or operation, and the principal pathological findings were thickening of the leaflets, commissural fusion, and large, obstructing thrombotic vegetations. Only 3 patients had annular abscess. Nine patients were treated surgically, and 6 died.

Our experience with prosthetic valve endocarditis is limited to 24 patients, and although it is a small series for analyzing the significance of clinical and pathological variables, an operative mortality of 12.5% (three deaths among 24 patients) appears to be a remarkable improvement in surgical results compared with results in previous reports [6, 8–11]. Our 24 patients were comparable with those in other series: 8 were in shock when operated on, and 18 (75%) had annular abscess. Our improved surgical results reflect an increased experience with valve reoperation, better operative techniques to manage patients with extensive destruction of the left ventricular inflow and outflow tracts due to abscesses [3–5], improved methods to control coagulation disorders, which are frequently encountered in these patients [10], and technological advances in the management of patients with multiorgan failure when they are transferred from the operating room to the intensive care unit. Our three operative deaths occurred in patients who were moribund and in shock before operation.

We believe that patients with mechanical valve endocarditis should undergo operation as soon as the diagnosis has been established. Endocarditis in bioprostheses can be managed more conservatively providing there is no echocardiographic evidence of valve dysfunction [10]. Regardless of the type of prosthetic valve, these patients should be hospitalized in institutions with open-heart surgical facilities, and a surgeon should be involved in the management of these patients from the time the diagnosis is being contemplated. This is probably the only way the mortality and morbidity will decrease in patients with prosthetic valve endocarditis.

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(For Discussion see page 712.)

Valve Repair in Acute Endocarditis

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Forty patients were operated on in the early phase of active endocarditis between 1980 and 1988. Indications for operation were heart failure (30 patients), severe valvular regurgitation (4), uncontrolled sepsis (2), septic emboli (3), and other (1 patient). Time between onset of endocarditis symptoms and operation ranged from 12 to 45 days (mean, 30 days). The aortic valve was involved in 3 patients; the mitral valve, in 28; both valves, in 7; and the tricuspid valve, in 2. There was no previous underlying valve pathology in 40%. Lesions found were cusp perforation (17 patients), annular abscess (4), vegetation (13), and chordal rupture (22). Positive blood cultures were found in 30 patients (75%). Bacterial findings were *Streptococcus* in 12 patients (30%), *Staphylococcus* in 15 (37.5%), gram-negative in 3 (7.5%), and unknown in 10 (25%). Criteria to perform valve repair were adequate antibiotic therapy for at least 1 week and large excision of all macroscopically involved tissues. In all cases, Carpentier's reconstructive techniques were used. Perioperative mortality was 2.5% (1 patient). Reoperation was necessary in 1 patient. Late mortality was 2.5% (1 patient). Repair was assessed either by angiography or by Doppler echocardiography before hospital discharge: 32 patients showed no regurgitation, whereas 7 had mild regurgitation (3 aortic, 4 mitral). Mean follow-up of 30 months was achieved in all survivors. There was no recurrence of endocarditis and no reoperation for valvular insufficiency. We conclude that valve repair in acute endocarditis is possible and effective in most instances.

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Although wide use of antibiotics has modified the course of acute bacterial endocarditis, some patients still require early operation. At Broussais Hospital, more than 6,000 mitral valve repairs have been performed according to Carpentier's techniques in a large range of pathology, including valve endocarditis. Because of our good results in subacute endocarditis, we have progressively extended our indications to acute bacterial endocarditis, that is, in patients operated on within the first 6 weeks after onset of symptoms and before completion of the initial full course of antibiotics. We therefore report our experience in 40 patients with special emphasis on surgical techniques and present long-term follow-up of valvular repair in acute endocarditis.

Material and Methods

Patient Population

Between January 1980 and December 1988, 40 patients with acute native valve endocarditis underwent valve repair at Broussais Hospital. Operation was indicated before the initial full course of antibiotics was completed for one or several of the following reasons: hemodynamic deterioration (n = 37), persistent sepsis (n = 4), or mobile vegetations at echocardiography with or without previous emboli (n = 6). We excluded from this study patients operated on more than 6 weeks after the beginning of antibiotic therapy who all had negative blood cultures and sterilized valve specimens.

Our policy has changed through the years. In the first 19 patients, we required at least a 2-week course of antibiotics and negative blood cultures at the time of surgery. In a second series of 21 patients, these criteria were not judged necessary and patients with less than 1 week of appropriate medical therapy and positive blood cultures were accepted for operation.

Patients ranged in age from 2 to 70 years (mean age, 33 years). There were 25 male and 15 female patients. Valves involved are summarized in Table 1. In 25 patients, the mitral valve alone was infected, and in 7 patients the infectious process affected both the mitral and the aortic valves, in 1 of them with a mitroaortic dislocation.

Preexisting Valve Pathology

Underlying valve pathology was present in 60% of the patients (Table 2). The origin was rheumatic in 11 patients, degenerative in 5, congenital in 6, and traumatic in 1 patient.

Origin of Infection

Predisposing factors were found in only 50% of patients. Dental manipulation was the most common potential

Table 1. Location of Acute Endocarditis

Valve	No. of Patients
Mitral	35
Isolated	25
Mitroaortic	7
Mitrotricuspid	3
Aortic	3
Tricuspid	2

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Table 2. Underlying Valve Pathology

Valve	Rheumatic	Congenital	Degenerative	Traumatic
Mitral	9	3	3	1
Aortic	4	1	2	0
Tricuspid	0	2	0	0
Total	13	6	6	1

source of infection (11 patients). The other sources of infection were gynecological (2 patients), meningitis (2 patients), drug addiction (1 patient), infection of an arteriovenous fistula for hemodialysis (1 patient), colic (1 patient), respiratory (1 patient), and infection of a central venous line in a patient in the intensive care unit.

Preoperative Bacteriological Findings

The microorganisms responsible for the infection were isolated in blood culture in 30 patients before operation (Table 3). *Staphylococcus* was the most frequent organism (37.5%), followed by *Streptococcus*, especially the *viridans* subgroup (30%), and gram-negative organisms (7.5%). In the remaining 25% of patients, no organism could be identified. It is noteworthy that 21 patients (52.5%) were operated on with positive blood cultures. All patients received varying courses of antibiotics for three to 45 days. No patient had fungal endocarditis.

Indications for Operation and Surgical Findings

Indications for operation were either hemodynamic (37 patients), bacteriological (4 patients), or echocardiographic (6 patients). Seven patients with either a bacteriological or an echocardiographic indication also had hemodynamic indication. Hemodynamic indications comprised dyspnea, hypoxia, and marked pulmonary edema on chest roentgenograms. Nine patients (23%) had a low cardiac output status (cardiac index <2 L/min/m^2). Bacteriological indications were uncontrolled sepsis in 4 patients. All patients had a fever above 39°C and had had antibiotic therapy for at least five days. The organisms involved were *Staphylococcus aureus* (n = 2), *Streptococcus* (n = 1), and *Hemophilus* (n = 1). Echocardiographic indications were based on the presence of mobile vegetations appended on the atrial side of the mitral valve (Fig 1). Five of these patients experienced previous systemic emboli: two cerebral and three peripheral (two lower limbs and one splenic).

Table 3. Preoperative Bacteriology

Microorganism	Patients n (%)
Staphylococcus	15 (37.5)
aureus	9
epidermidis	6
Streptococcus	12 (30)
viridans	5
β-hemolytic	3
α-hemolytic	1
sanguis	1
Untyped	2
Gram-negative	3 (7.5)
Hemophilus	2
Bacillus	1
Unknown	10 (25)

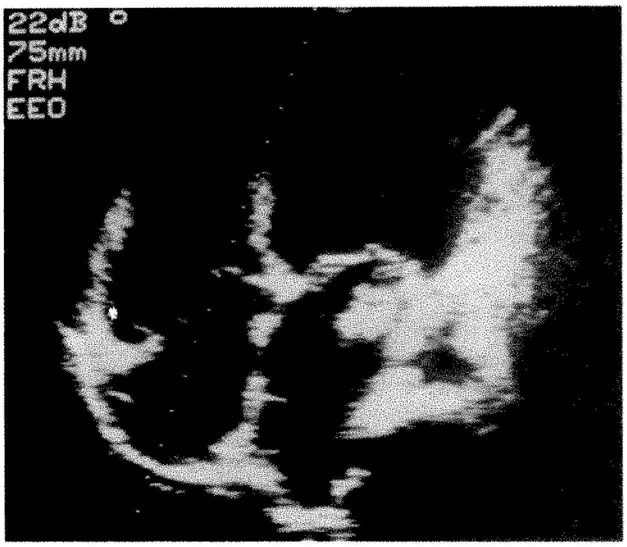

Fig 1. Echocardiography shows a mobile vegetation appended to the atrial aspect of the mitral valve.

Operative Findings

MITRAL LESIONS. The mitral valve findings are shown in Table 4. Chordal rupture was the predominant lesion, present on the anterior leaflet in 14 patients and on the posterior leaflet in 8 patients. Only marginal chordal rupture was taken into account as leading to valvular prolapse. Rupture involved more than one chorda in all instances. The paramedian chordae of the anterior leaflet were ruptured in 6 of the 14 patients.

Leaflet perforation was present in 13 patients, varying in size from 1 cm^2 to almost the entire anterior leaflet (Fig 2). Vegetations were present in 18 patients, extending to the atrial wall in 10 patients. Abscess was present in 5

Table 4. Mitral Valve Lesions at Operation

Lesions	No.
Chordal ruptures	22
Anterior leaflet	14
Posterior leaflet	8
Perforations	13
Anterior leaflet	8
Posterior leaflet	5
Vegetations	18
Abscess	5
Annular	2
Valvular	3

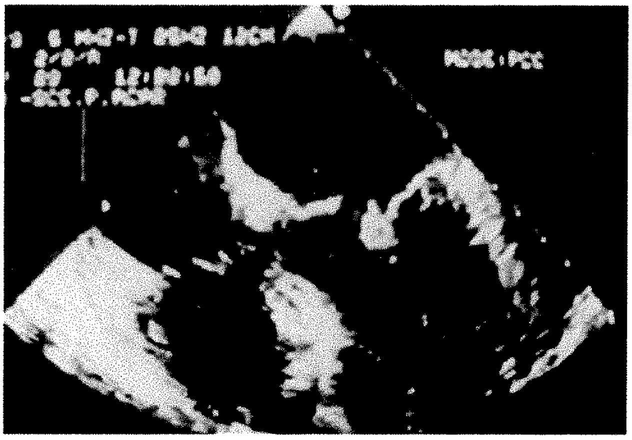

Fig 2. *Transesophageal echocardiogram shows a perforated anterior leaflet as well as a vegetation of the free edge.*

patients, involving the annulus (n = 2) or the valve tissue (n = 3). One patient had an extension to the subvalvular apparatus, reaching the anterior papillary muscle. In another patient, the abscess had split the mitroaortic junction, detaching the anterior leaflet of the mitral valve from the annulus.

AORTIC LESIONS. In 3 patients, the aortic valve alone was involved by the endocarditic process. Cusp perforations limited to one cusp were present in all patients leading to acute regurgitation. There was no annular abscess. Of the 7 patients with mitroaortic endocarditis, 5 had extensive lesions of the aortic valve as an abscess involving one cusp or more (n = 3) or an annular abscess (n = 2) with cusp destruction.

TRICUSPID LESIONS. The tricuspid valve alone was involved twice. Lesions were similar to those found in the mitral valve, namely, two chordal ruptures and one leaflet perforation. The tricuspid valve was involved with the mitral valve in 3 patients. Lesions were always chordal rupture (n = 3).

Intraoperative Bacteriology

All specimens were sent for culture and microscopic examination. Twelve valve specimen cultures were positive and three displayed only inflammatory cells when examined microscopically, although the cultures were negative. Blood culture and valve tissue cultures were discordant for *Staphylococcus aureus* (Table 5).

Table 5. Bacterial Findings at Operation

Organism	Blood culture+ Valve culture+	Blood culture+ Valve culture−	Blood culture− Valve culture+
Staphylococcus	6	2	5
Streptococcus	1	9	0
Gram-negative	0	3	0
Total	7	14	5

+ = positive; − = negative.

Table 6. Surgical Technique for the Mitral Valve

Technique	No.
Valvular repair	
Posterior leaflet resection	13
Anterior leaflet resection	4
Leaflet patching	7
Direct suture	2
Subvalvular repair	
Chordal shortening	3
Chordal transposition	8
Annular remodeling	
Commissural refection	7
Prosthetic ring	19

Operative Techniques

All operations were performed under cardiopulmonary bypass with either intermittent aortic cross-clamping at 21°C or moderate hypothermia at 28°C and crystalloid cardioplegia. The surgical principles were as follows: All macroscopically involved tissues were largely resected without any concern for the possibilities of repair. The valve was widely resected, including a strip of at least 2 mm of normal valvular tissue. Valve reconstruction was then performed according to Carpentier's techniques and principles. The procedures used are shown in Table 6.

Some specific techniques were used for acute valve endocarditis. Autologous pericardial patching was used for perforations of the anterior leaflet of the mitral valve (n = 7) and for cusp perforation of the aortic valve (n = 5) (Fig 3). Pericardium was tanned intraoperatively in a 0.6% glutaraldehyde solution for ten minutes and washed in a saline bath. The smooth surface of the pericardium was turned toward the atrium for mitral valve repair and toward the ventricle for aortic valve repair. The entire anterior leaflet of mitral valve was replaced with pericardium in 3 patients (Fig 4). This procedure was possible only when the marginal chordae were not involved by the infectious process. Otherwise, an associated chordal transposition was mandatory. Another specific technique used was chordal transposition [1] (n = 8) whenever marginal chordae of the anterior leaflet were ruptured. Marginal chordae of the posterior leaflet were selected, detached with a tiny part of the posterior leaflet, and then transposed onto the anterior leaflet in an adequate position (Fig 5). This type of posterior chordal transposition may be combined with a quadrangular resection depending on the extent of the transposition.

Commissural refection was performed in 7 patients. This technique was indicated when the commissural area of both anterior and posterior leaflets was prolapsed owing to ruptured commissural chordae. A commissural reconstruction by leaflet sliding plasty [2] was preferred to the previously used commissural resection to avoid leaflet distortion secondary to a large annulus plication (Fig 6). In this technique, when all infected tissues are removed, the commissural area of the anterior and posterior leaflet is

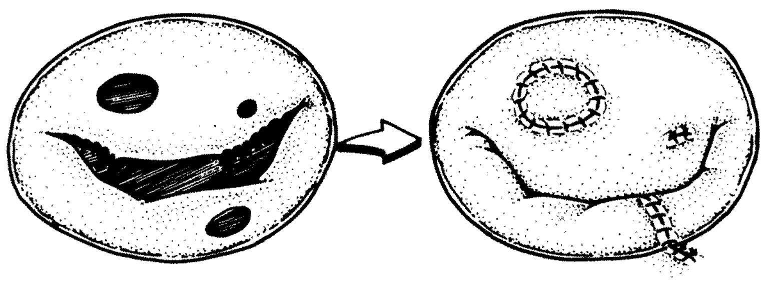

Fig 3. Leaflet perforation treated by pericardial patch closure (anterior leaflet) and quadrangular resection (posterior leaflet).

detached from its annular attachment to approximate the two leaflet remnants, which are then sutured together.

Carpentier's prosthetic ring was necessary in only 19 patients. Use of the ring was indicated when the annulus was dilated by a previous underlying pathology, particularly in degenerative lesions, or for reinforcement of annular remodeling after an extensive quadrangular resection.

Aortic valve repair was always reinforced by an annular running 2-0 suture to avoid diastolic distention, and cusp perforation was patched with pericardium (n = 5). Tricuspid valve repair included chordal transposition (n = 5) and leaflet patching (n = 1). It was always associated with a ring annuloplasty.

Associated Procedures

Aortic valve replacement was necessary in 5 patients (three bioprotheses and two mechanical prostheses). One patient with mitroaortic endocarditis underwent aortic valve repair and mitral valve replacement because of an extensive abscess formation including the posterior papillary muscle. One patient with partial atrioventricular septal defect underwent repair based on the trileaflet concept. An atrial septal defect was closed with autologous pericardium once. The patient with mitroaortic dislocation underwent pericardial patching between the aortic annulus and the mitral annulus. An aortic valve replacement was performed with a Carpentier-Edwards bioprosthesis, and the anterior leaflet of the mitral valve was sutured to the pericardial patch and then reinforced by a prosthetic ring.

Results

Hospital Mortality

There was one hospital death (2.5%). This patient underwent extensive patching of the anterior leaflet of the mitral valve; although the valve was fully competent, he died of persistent sepsis leading to cardiogenic shock with low systemic resistance caused by "bacterial switch." The primary organism was *S aureus*, which changed to a multiresistant *Pseudomonas* in the postoperative intensive care unit.

Reoperation

On postoperative day 10, 1 patient, a 5-year-old boy, required emergent reoperation after mitral valvoplasty. He had ruptured chordae of the anterior leaflet, vegetations on the posterior leaflet, and congenital aortic stenosis. After aortic commissurotomy, mitral valve repair consisted of chordal transposition and quadrangular resection of the posterior leaflet. At reoperation, transposed chordae and suture of the quadrangular resection were partially torn. A new valvoplasty, with a larger excision of the altered tissues, was performed with new chordal transposition and a pericardial patching of the posterior leaflet to avoid excess traction on the suture lines. The postoperative course was then uneventful.

Complications

Postoperative low cardiac output in 1 patient was successfully treated by intraaortic balloon. A transient atrioventricular block occurred in 3 patients, but did not require placement of a permanent pacemaker.

Early Results

Immediate hemodynamic results have shown a dramatic improvement in clinical congestive heart failure in all survivors. Continuous antibiotic therapy was prolonged for up to 2 months with adaptation to the germ isolated in valve cultures.

Valve repair was assessed in all survivors before hospital discharge, either by Doppler echocardiography (n = 36) or by angiography (n = 3). Thirty-two patients showed no regurgitation, whereas 7 had mild regurgitation (3 aortic and 4 mitral) without hemodynamic significance.

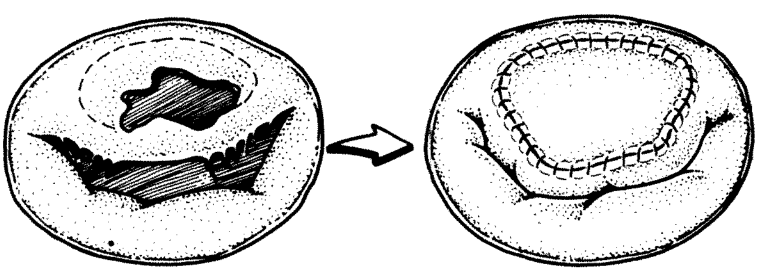

Fig 4. Pericardial patch replacement of almost the entire anterior leaflet.

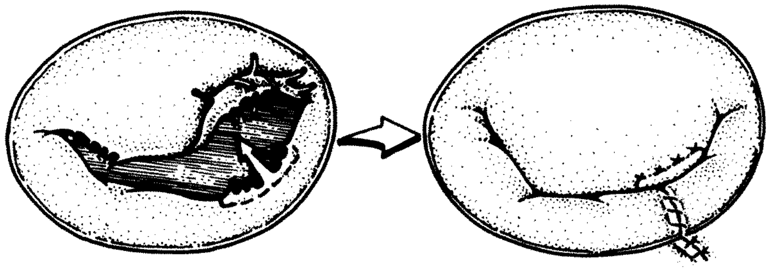

Fig 5. Chordal transposition from the posterior leaflet to the free edge of the anterior leaflet.

Late Follow-up

Late follow-up was available in all patients. There was one late death, 2.5 years after repair, in a 2-year-old boy with a hammock valve in whom the repair was satisfactory. Postmortem examination was not available. Mean follow-up was 30 months (range, 6 to 94 months). No recurrent endocarditis was observed, and no reoperations were required.

Comment

Early operation for acute endocarditis that does not respond fully to medical therapy is now widely accepted [3–6]. Although heart failure has become the most common indication, it sometimes is underestimated [7], and operation therefore is delayed, leading to a 10% to 30% postoperative mortality [5–10]. As do Becker and colleagues [11] and Wilson and co-workers [12], we believe that progression of heart failure in patients with mitral endocarditis may be as rapid as aortic endocarditis and that such patients should be operated on as soon as possible. Consequently, our policy is much more aggressive than that of other investigators. We also believe, as do Lewis and associates [13], that mortality in mitral endocarditis could be lowered if patients were operated on soon after admission. Our hospital mortality is 2.8% which is, to our knowledge, one of the lowest ever reported in such conditions.

The organisms involved, especially *Staphylococcus* or gram-negative organisms, are often responsible for extensive lesions beyond the valve itself, as annular abscess or septic aneurysms, despite adequate antibiotic therapy. The endocarditic process usually begins on the valve leaflet where it remains located for some time [4, 14]. For all these reasons early operation is recommended [11, 12]. Until now, however, acute bacterial endocarditis has been considered a contraindication to valve repair. Our results show that not only is valve repair feasible, but it is also safer than valve replacement at the early stage as it avoids use of prosthetic material. Valve repair strongly correlated to the lack of recurrence of infection in our series in contrast to previous experiences with prosthetic valve replacement, in which the rate of infection reached 8% to 20% [4, 9, 15]. Sweeney and associates [15] reported that all reinfections involved the same organism cultured at the initial valve replacement. Although valvular replacement has dramatically improved acute endocarditis outcome, the rationale of inserting any prosthetic material in an infected site remains uncertain. We also stress that if valve reconstruction is safe in the early phase of acute endocarditis, it has much better results in the long-term than any prosthetic valve replacement [16].

When both mitral and aortic valves were involved, we attempted to repair the mitral valve, even when the aortic valve was replaced. This attitude is well documented by the case of mitroaortic dislocation in which a pericardial patch allowed us to repair the mitral valve. Long-term survival rate, thromboembolic rate, and reoperation rate are significantly different for patients who have two prosthetic valves as opposed to those who have one prosthetic valve [16]. Other investigators have shown that left ventricular function is preserved when the subvalvular apparatus is not transected, which always favors mitral valve repair.

In a first step, we performed repair only in patients who had had at least 2 weeks of antibiotic therapy and negative blood cultures (n = 19). In a second step, according to our early results, 21 patients (52.5%) with positive blood cultures and less than 1 week of antibiotic therapy were accepted for operation. Early repair has several advantages: (1) it avoids extension of the infecting process; in our experience, there were only two annular abscesses and one mitroaortic disruption; (2) it preserves the subvalvular apparatus and therefore prevents impairment of left ventricular function secondary to valve replacement; and (3) it leaves tissues that are more resistant to infection than prosthetic material; for this reason, we avoided use of a prosthetic ring as often as possible. We had only one

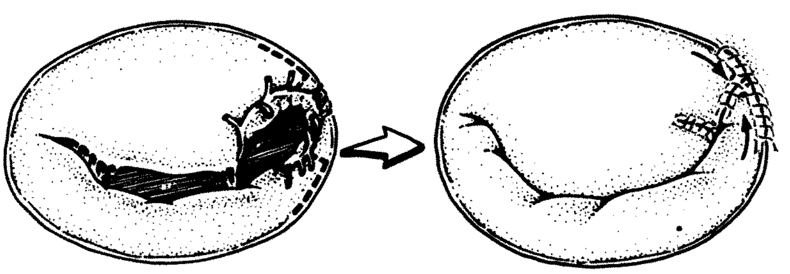

Fig 6. Sliding commissuroplasty for a prolapse of both anterior and posterior leaflets in the paracommissural area.

postoperative death caused by persistent uncontrolled sepsis, and one reoperation on postoperative day 10 for acute recurrent mitral regurgitation without recurrent infection. Another repair was possible, however, and the postoperative course was then uneventful. Scattered reports, such as those by Fleischer and colleagues or Hugues and Noble [17, 18], have described successful conservative operation in acute bacterial endocarditis, but these investigators made no systematic attempt to repair rather than to replace infected valves during the acute phase of endocarditis.

We believe that the organisms involved must not influence surgical policy. Thirty-seven percent of our patients had staphylococcal infection, and 7.5% had gram-negative infection. Although this study is retrospective, we could not show any statistical difference in terms of outcome regarding the organism involved, as no patient experienced recurrent endocarditis, and none needed reoperation in the long term. Our population did not include fungal endocarditis, and only 1 patient was a drug addict. Fungi sometimes cannot be seen macroscopically, leading to incomplete debridement and thus unreliable valve repair. We therefore conclude that valve repair in acute endocarditis is as safe as repair for any other valve disease, especially for the mitral valve.

If repair is effective in acute endocarditis, the only limiting factor is the extent of the lesions. As all infected tissues must be resected, it is easy to imagine patients in whom not enough valve tissue or subvalvular apparatus is left to allow valve repair. Development of new techniques, however, has permitted us to enlarge our indications, and during the past 2 years less than 20% of our patients with acute bacterial endocarditis required valve replacement in the mitral position. Use of a prosthetic ring in 19 patients in our series was necessitated mainly by a preexisting mitral lesion. In chronic mitral insufficiency, prosthetic ring annuloplasty is mandatory because of annular dilatation or deformation subsequent to correction of leaflet prolapse or subvalvular anomaly [1, 16]. Therefore, when chronic valve lesions are present before acute endocarditis, use of a prosthetic ring is necessary. In acute mitral regurgitation secondary to endocarditis, no underlying pathology exists; therefore, no annular dilatation or deformation exists. For this reason, no prosthetic ring was necessary in 16 patients. The implantation of a ring does not increase the risk of infection, however, because acute bacterial endocarditis usually spares the annulus.

Another question of great matter is whether an isolated single vegetation with no or minimal valve incompetence and no previous systemic emboli requires an operation. We have operated on 1 patient with this condition and strongly believe that it is better to operate because lesions are time dependent. Operation avoids the risk of infectious extension.

Finally, the age of the patients was not a limiting factor in reconstructive operation, as our patients ranged in age from 2 to 70 years.

In summary, although most acute valve endocarditis can be effectively controlled with a 6-week course of antibiotic therapy, some patients still require early operation for hemodynamic, bacteriological, or echocardiographic reasons. Repair is possible in most patients, with results that appear to be superior to those of valve replacement.

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DISCUSSION

(Papers by David [page 701] and Dreyfus [page 706] and their associates)

DR AUBREY C. GALLOWAY (New York, NY): I enjoyed the presentations of both Dr David and Dr Dreyfus immensely. Our experience at New York University and Bellevue Hospital probably more closely parallels that presented by Dr David, although we have performed valve repair in select cases of acute endocarditis.

At Bellevue Hospital, most of the patients we see are extremely sick with a very aggressive form of endocarditis, and sepsis is one of the major indications for operation. I think this group with overwhelming sepsis is very dissimilar to the group discussed by Dr Dreyfus. In patients with extreme sepsis, valve replacement is usually needed because the valves are massively destroyed.

However, I was somewhat surprised by the large need for patch reconstruction of the outflow tract and annulus in the series of David and associates. In general, we have employed this technique in less than 10% of our patients; usually it is possible to resect the annular abscess and close any annular defect primarily with a figure-of-8 suture of 4-0 Prolene. We then bridge that closure with pledgeted valve sutures. That method has been successful in most instances, and we patch only when there is a ventricular septal defect or when a massive abscess extends outside the heart. Dr David, what are your indications for placing a patch? Patch placement in 60% to 70% of patients is much higher than we would expect.

In regard to the excellent paper of Dreyfus and associates, we agree with the premise that valve reconstruction is possible in a very select group of patients with mitral valve endocarditis. Out of more than 360 mitral valve repairs, 14 reconstructions were performed in the acute phase of endocarditis and a similar number late after endocarditis. This is out of more than 150 patients with acute endocarditis operated on during that period. Valve reconstruction was done between day 1 and day 16 in the acute phase of endocarditis. There were no deaths in this select group. However, this group is markedly different from the sicker, more septic group reported by David and associates.

In patients undergoing valve repair in our series, we primarily did a rectangular resection of the infected posterior leaflet, although in a few, we closed small perforations directly in the anterior leaflet. In 1 patient, we performed a pericardial patch repair of the anterior leaflet. In the presence of ruptured chordae to the anterior leaflet, we prefer to transpose a secondary chord and resuspend the leaflet to the secondary chord.

All of our patients who underwent valve reconstruction have received late echocardiographic follow-up. There has been no recurrent endocarditis and no reoperation in this small subgroup. One patient had major mitral insufficiency on the echocardiogram. We operated on 3 patients who have continued intravenous drug abuse for longer than 2 to 3 years, and recurrent endocarditis has not developed. Thus, valve repair appears relatively resistant to recurrent endocarditis compared with valve replacement.

However, our patients and the patients of Dreyfus and associates appear to be a small subgroup of patients with infection who require mitral valve operation. To apply valve reconstruction, isolated, very discrete infection or abscess should be present. Otherwise, with extensive infection, valve replacement is still the procedure of choice.

Dr Dreyfus, when do you abandon valve reconstruction and perform valve replacement?

DR SIDNEY LEVITSKY (Boston, MA): I am going to confine my remarks to the presentation of Dr David, which involved a very carefully performed clinical study that carried several important messages. In addition, because of the different social mix in the selection of patients, our experience with the operative treatment of acute endocarditis at the University of Illinois Medical Center from 1980 to 1988 presents a slightly different emphasis. During those years, a total of 133 patients underwent operation for acute endocarditis. The majority, namely, 117 patients, had native valve endocarditis. Anatomically, the tricuspid valve was involved in 27, the mitral valve in 43, the aortic valve in 46, and the aortic and mitral valves in 17. Of additional importance, 98 of the 133 patients were intravenous drug abusers. The hospital mortality for the entire group was 7.4%.

There was an equal incidence of aortic and mitral valve endocarditis, which is contrary not only to Dr David's experience but also to the great preponderance of reports in the literature, which usually have a higher rate of aortic valve involvement. In addition, we had a much higher incidence of gram-negative sepsis, mostly *Pseudomonas*, which we encountered in 27% of our patients as opposed to only 3% of the patients in the series of David and associates. All of these differences could be attributed to the high incidence of intravenous drug abusers in our series.

I have two questions for Dr David. One involves the fate of the pericardial patch. In most instances, we have been able to reconstruct the left ventricular outflow tract using local tissue rather than foreign material or a pericardial patch. We had poor experience in the past, particularly in patients with *Pseudomonas* infection, using pericardium to reconstruct the valve annulus, especially when either a periannular or ventricular abscess was present. Second, in our large group of patients we have noticed a high incidence of postoperative bleeding, probably related to acute sepsis. Over the past 3 years, using protocol approved by the Food and Drug Administration with Immuno's fibrin sealant in spray form, we have been able to significantly decrease the incidence of postoperative bleeding. Because fibrin sealant is available in Toronto, I wonder whether Dr David advocates using this material for this group of patients.

Again, I enjoyed both of these presentations on endocarditis. I believe this is a particularly important and relevant topic for cardiac surgeons in the 1990s as we become more aggressive about operating on patients with endocarditis at an earlier period in the course of the disease so as to avoid the dreaded complications of periannular and myocardial abscess.

DR WILLIAM W. ANGELL (La Jolla, CA): My colleagues and I agree with mitral valve repair in patients with bacterial endocarditis when it can be accomplished. We have done this in only 4 of 207 mitral valve repairs but have applied all the techniques described, including ring annuloplasty and leaflet switch. A notable difference in our experience compared with that of Dreyfus and associates is the mean age of the patients, 71 years and 32 years, respectively.

One of my questions involves the indication for valve replacement in patients seen with acute endocarditis. I wonder whether the chance of being able to repair these valves is not substantially enhanced by waiting for 4 to 6 weeks after the acute phase of the illness.

Another question concerns the long-term follow-up when the aggressive approach with the patch techniques that have been described is used. Our general experience is that with a porcine xenograft, 8 to 10 years of valve function is typical, particularly in the elderly population.

We have had success with patients whose cultures are negative for endocarditis by withholding all antibiotic therapy for several days before operation and waiting until after the tissue specimens have been retrieved at operation to give any antibiotics. Positive cultures result.

In my opinion, for a destroyed aortic root, particularly with prosthetic valve endocarditis, the homograft valve is still the replacement of choice, especially when a conduit is required. It solves many of the reconstructive problems associated with annulus destruction.

DR TIRONE E. DAVID (Toronto, Ont, Canada): Dr Galloway asked what are our indications for patch reconstruction of the annulus in patients with endocarditis. We have such an experience with patch enlargement of the aortic annulus in patients undergoing aortic valve replacement with a bioprosthesis (in an attempt to match the size of the prosthesis to the size of the patient) that we tend to widely excise any aortic annular abscess and reconstruct the aortic root with autologous pericardium. Thus, except in patients with aortic annular abscess smaller than 2 to 3 mm in whom debridement and primary closure of the cavity is easily accomplished, we reconstruct the outflow tract with pericardium. A similar approach is used in patients with mitral annulus abscess.

As far as the incidence of abscess is concerned, the literature on this subject indicates that all patients with mechanical valve endocarditis and many with bioprostheses have periannular infection at the time of operation or autopsy. The incidence of annular abscess in our patients with native or prosthetic valve endocarditis is similar to that in series previously published.

Dr Levitsky, I agree that patients with infective endocarditis in Toronto are probably different from those in Chicago. Among our 62 patients, only 5 were intravenous drug users, whereas the majority of your patients were intravenous drug users. This fact alone would explain the differences in microorganisms as well as the heart valve that is more commonly infected.

As for autologous pericardium as patch material, we have used it for aortic annulus enlargement in several hundred patients during the past decade without a single case of false aneurysm. I have reoperated on a number of these patients because of failed bioprostheses, and the patch was always intact. The pericardial patch becomes thicker, and sometimes it is difficult to identify where it was placed. In 1984, we began to reconstruct the mitral annulus with autologous pericardium, and to date we have performed 37 such procedures for endocarditis, for extensive calcification of the mitral annulus, or for mitral valve rereplacement. We have not yet had a single case of valve dehiscence. When autologous pericardium is not readily available, we have used glutaraldehyde-preserved bovine pericardium with equally good results.

Dr Angell, I agree that a homograft is likely the best graft to implant in the infected aortic root, and we did use it in a few patients. My main difficulty with homografts is procurement. Our demand far exceeds our supply; I always have many more patients awaiting an aortic homograft than homografts I can harvest.

DR DREYFUS: Concerning Dr Galloway's remarks, I just want to make a comment about the secondary chordae transposition of the anterior leaflet to the free margin. Those chordae are often too short or too long and do not fulfill the criteria of reference points; you sometimes see secondary leaflet restrictive motion or leaflet prolapse. Our indications, as he asked, are very wide. As I stressed, probably the organism involved, as fungi or gram-negative, would lead us not to perform valvular reconstruction in certain instances. The only instances in which we cannot try to perform valve reconstruction are cases of papillary muscle abscess, because in these cases we cannot be sure of the subvalvular apparatus.

At Broussais, in the beginning we would replace 80% of these valves and then treat with reconstruction 20%. We have switched to the opposite. Now we repair 80% and probably replace only 20%.

Dr Angell, our indication for replacement, as I said previously, is the total destruction of the subvalvular apparatus. I do not believe there is any rationale to replace a mitral valve because there is an abscessed annulus. I mean, you really enclose an abscess with a prosthesis more than you would do with a reconstruction.

Now, concerning the fate of waiting more than 6 weeks, we fully agree that if you wait more than 6 weeks you would have healed lesions and that probably operation would be the same as for any other mitral valve disease. As you know, however, hemodynamics is one of the main determinants of poor outcome, and we think that the mitral valve, as the aortic valve, can decompensate very quickly.

Concerning the pericardial patch, I agree with Dr David in that we have reoperated on patients, especially with a pericardial patch on the ventricular septal defects, and we have not found any shrinking or any other modification or calcifications.

Intraoperative Management of Patients With Heparin-Induced Thrombocytopenia

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For 11 patients with confirmed heparin-induced thrombocytopenia, we used reversible platelet inhibition with iloprost, a stable prostacyclin analogue, to permit safe heparin administration for cardiac (n = 9) or vascular (n = 2) operations. In vitro, iloprost (0.01 μmol/L) prevented both heparin-induced platelet aggregation and ^{14}C-serotonin release in all patients. Therefore, intraoperatively, a continuous infusion of iloprost was started before administration of heparin and was continued until 15 minutes after administration of protamine. For cardiac patients, after heparin administration, the whole blood platelet count did not change (171,000 ± 29,000/μL versus 174,000 ± 29,000/μL, mean ± standard error of the mean); no spontaneous platelet aggregation was observed, and plasma levels of the α-granule constituents platelet factor 4 and β-thromboglobulin increased from 38 ± 14 and 140 ± 18 ng/mL to 591 ± 135 and 235 ± 48 ng/mL, respectively. Fibrinopeptide A levels actually decreased from 287 ± 150 to 27 ± 6 ng/mL. Furthermore, adenosine diphosphate-induced platelet activation was preserved, postoperative bleeding times were unchanged, and no heparin-related deaths occurred. Similar results were obtained in both vascular patients. We conclude that temporary platelet inhibition with iloprost now permits safe heparin administration in all patients with heparin-induced thrombocytopenia who require a cardiac or vascular operation.

(Ann Thorac Surg 1990;49:714–23)

Use of heparin as an effective yet reversible anticoagulant has contributed to advances in both cardiac and vascular surgery. Soon after its clinical introduction in 1933, however, potentially detrimental interactions between heparin and platelets were recognized. A transient mild decrease in the circulatory platelet count was first noted in mice and dogs in 1941 and has since been recognized as a nearly universal occurrence in humans [1, 2]. This mild platelet "agglutination" must be distinguished from a severe thrombocytopenia associated with thrombosis that can also complicate heparin therapy. This phenomenon has been termed heparin-associated or heparin-induced thrombocytopenia (HIT) and is believed to be immune mediated [3, 4].

Because of an overall morbidity and mortality reported to be as high as 61% and 23%, respectively, avoidance of all heparin-containing products including heparin flush and even heparin-coated catheters is mandatory once the diagnosis of HIT is established [3, 5]. For patients with confirmed HIT who require urgent cardiac or vascular operations, however, reexposure to heparin is unavoidable. Because the adverse consequences of heparin administration are mediated largely through heparin-induced platelet activation, we evaluated the concept that temporary inhibition of platelet function during heparin exposure would reduce or eliminate any proclivity for heparin-associated thrombosis. We evaluated, in vitro, the ability of iloprost (ZK 36374), a prostacyclin analogue, to prevent heparin-induced platelet activation, comparing its efficacy with that of "conventional" platelet inhibition with aspirin. Iloprost was then administered to 11 patients during operation. We outline a management approach for patients with confirmed HIT and report our experience using iloprost to prevent heparin-induced thrombosis during cardiac and vascular operations.

Material and Methods

Patient Population

Eleven patients with confirmed HIT required either cardiac (n = 9) or vascular operations (n = 2). The clinical diagnosis of HIT was entertained in these patients after thrombotic or untoward hemodynamic events associated with heparin exposure (Table 1). Furthermore, for all patients in vitro heparin-induced platelet activation was again documented before the time of operation, confirming the diagnosis (discussed in Results section under *Laboratory Diagnosis*) [6]. For all patients, the conduct of operation and anesthesia did not differ from similar cardiac or vascular procedures performed at the Hospital of the University of Pennsylvania. All cardiac operations were performed by Dr Addonizio at the Hospital of the University of Pennsylvania except that of patient 2, whose operation was performed at the National Institutes of

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Table 1. Clinical Course

Patient	Age (yr)	Complications Associated With Heparin Administration	Indication for Reexposure to Heparin	Maximum Dose of Iloprost (ng/kg/min)	Perioperative Complications
1	41	Deep venous thrombosis, paradoxical emboli/CVA	Atrial septal defect, tricuspid valve replacement	10	None
2	64	Respiratory insufficiency, multiple pulmonary emboli (probable)	Aortic/mitral valve replacement	30	None
3	55	Unstable angina/myocardial infarction	CABG	30	None
4	69	Unstable angina/cardiac arrest	CABG	24	None
5	68	Cardiac ischemia	CABG	48	None
6	69	Spontaneous platelet aggregation, cardiac arrest	CABG	24	None
7	75	Unstable angina	CABG	24	Ventricular fibrillation on postoperative day 3
8	51	Thrombosis of bifemoral artery, pulmonary emboli, gastrointestinal bleeding	CABG	24	None
9	72	Thrombosis of retinal artery	Aortic valve replacement	36	None
10	70	Thrombosis of femoral artery	Carotid endarterectomy	24	None
11	76	CVA, thrombosis of subclavian vein	Aortic bypass graft	36	None

CABG = coronary artery bypass grafting; CVA = cerebrovascular accident.

Health.* All studies were approved by the Hospital of the University of Pennsylvania Institutional Review Board (patients 1, 3–9, and 12), the Human Research Committee of the National Institutes of Health (patient 2), and the Veterans Administration Human Experimentation Committee (patient 10). In addition, protocols were reviewed by the Food and Drug Administration before initiation of the clinical trial. Detailed verbal and written informed consent of each patient was obtained before operation by Dr Addonizio.

For all cardiac procedures, a membrane oxygenator system was used, consisting of either a Sci-Med oxygenator (Sci-Med Life Systems, Minneapolis, MN) or a Terumo oxygenator (Terumo Corp, Piscataway, NJ). The pump oxygenation system was filled (primed) with 2,000 mL of Normosol solution (Abbott Laboratories, Chicago, IL) to which had been added 200 mL of a 25% albumin solution and 5,000 U of porcine mucosa heparin according to our standard routine.

As a frame of reference, for the cardiac patients, all preoperative, intraoperative, and postoperative variables were subjectively compared with the same variables measured in a group of 21 control subjects undergoing elective coronary revascularization. These patients were 21 consecutive men between the ages of 37 and 68 years (mean age, 59 years) who had coronary artery disease (mean bypass time, 97 minutes) but few risk factors for operation. In addition, this group also had normal hematological profiles preoperatively with no evidence of heparin-induced platelet activation [7, 8]. Where appropriate, for ease of comparison, data from these control patients are included in figures showing results from patients with HIT.

Blood Preparation

All blood samples for preoperative, intraoperative, and postoperative studies were drawn into plastic syringes containing one volume of 3.8% sodium citrate for every nine volumes of blood. All samples for platelet count, aggregometry, and serotonin release were analyzed immediately. Plasma samples for platelet factor 4 (PF4, platelet α-granule constituent; Abbott), β-thromboglobulin (β-TG, platelet α-granule constituent; Amersham, Arlington Heights, IL), thromboxane B_2 (stable end product of thromboxane A_2; New England Nuclear, Boston, MA), fibrinopeptide A (FPA, product of thrombin acting on fibrinogen; ByK Sangtec Diagnostica, FRG), and plasma levels of iloprost, although processed immediately, were frozen at −70°C for later analysis [9–13].

Platelet-rich plasma and platelet-poor plasma were prepared as previously described [14]. Platelets were counted by either phase microscopy or by Coulter Counter [15]. Platelet aggregation was performed according to the method of Carvalho and colleagues [16]. We considered complete second-wave aggregation for human platelets to be greater than 63% light transmittance when platelets were challenged by adenosine diphosphate (ADP) as previously described [17]. The threshold concentration of aggregating agent was defined as the lowest logarithmic concentration of aggregating agent required to produce complete second-wave aggregation. To obtain these thresholds, concentrations of ADP as great as 50 μmol/L were used [17].

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* A case report of patient 2 was presented to the American College of Surgeons (Surg Forum 1985;36:285–6).

Laboratory Diagnosis

To diagnose heparin-dependent platelet-activating factor, we added porcine mucosa heparin (0.1 to 3 U/mL) to a mixture of aspirin-free normal donor's platelet-rich plasma (300 μL) and the patient's platelet-poor plasma (200 μL). This assessment was repeated with the patient's own platelet-rich plasma (500 μL) when the circulating platelet count permitted preparation of platelet-rich plasma with a platelet count greater than 100,000/μL. After heparin was added, at least 20% aggregation or 6% ^{14}C-serotonin release, or both, within 45 minutes was considered positive [6, 18, 19]. Platelets were also tested against 5 or 10 μmol/L of ADP to confirm reactivity within the normal range. All patients met these criteria, thereby confirming the diagnosis of HIT. To evaluate their efficacy in preventing heparin-induced platelet activation, either iloprost (0.01 μmol/L in saline solution; Berlex Laboratories, Wayne, NJ) or aspirin (see Comment section; 4 mmol/L in Tris buffer, pH 8.6; Sigma Chemical, St. Louis, MO) was added to the aggregometry cuvette two minutes before heparin administration.

Infusion Protocol

Iloprost was prepared on the day of the study but not more than four hours before its intended use. Three ampules (each containing 0.1 mg/mL) were added to 297 mL of sterile saline solution for a drug concentration of 1,000 ng/mL.

The iloprost infusion was started immediately after induction of anesthesia. Infusion rates were increased incrementally with hemodynamic monitoring until prevention of heparin-induced platelet activation was achieved in vitro. This corresponded to infusion rates of 10 to 48 ng/kg/min. At that time, porcine mucosa heparin (100 to 300 U/kg) was administered and the operative procedure was started. At completion of the procedure, the iloprost infusion was tapered to 6 ng/kg/min. Protamine sulfate, which forms a complex with heparin that is cleared by the reticuloendothelial system, was then administered and the iloprost infusion was discontinued.

Blood Acquisition

All blood samples obtained during operation were drawn from indwelling arterial vascular catheters or from the heart-lung machine during cardiopulmonary bypass. Blood samples for platelet functional studies and measurement of platelet counts, hematocrit values, and PF4, β-TG, FPA, and thromboxane B_2 levels were obtained at the following times: after induction of anesthesia; at 15-minute intervals after the start of the iloprost infusion, before administration of heparin; 5 minutes after heparin administration; before protamine infusion; 15 minutes after protamine infusion; one and three hours after the patient's arrival in the surgical intensive care unit; and on the first postoperative day. Template bleeding times were performed after induction of anesthesia and again three hours after the patient's arrival in the surgical intensive care unit. Chest tube drainage was measured at six and 12 hours.

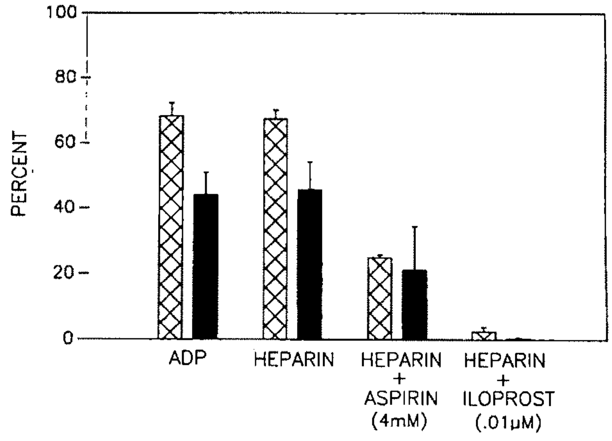


Fig 1. In vitro percentage of platelet aggregation (hatched bar) *and ^{14}C-serotonin release* (solid bar) *(mean ± standard error of the mean) in response to adenosine diphosphate* (ADP)*, heparin (1 to 3 U/mL), heparin plus aspirin (4 mmol/L), and heparin + iloprost (0.01 μmol/L).*

Results

Preoperative Diagnosis and Assessment

All patients had greater than 20% platelet aggregation and 6% ^{14}C-serotonin release in response to heparin (0.1 U to 3 U/mL) (Fig 1). Because the plasma level of heparin achieved during cardiac and vascular operations is 3 and 1 U/mL, respectively, these concentrations were used to test the in vitro efficacy of aspirin and iloprost in preventing heparin-induced platelet aggregation and release. Iloprost (0.01 μmol/L) prevented both in vitro heparin-induced platelet aggregation and ^{14}C-serotonin release in all patients. In contrast, the efficacy of aspirin (4 mmol/L) was quite variable; aspirin prevented aggregation and release in only 3 patients (Fig 1).

Perioperative Clinical Course and Assessment

All patients remained hemodynamically stable throughout their procedures. Although iloprost is a vasodilator, blood pressure was readily controlled with phenylephrine hydrochloride (Neo-Synephrine) (Table 2).

Platelet Counts

In the patients undergoing cardiac operations (n = 9), the mean whole blood platelet count did not change with heparin administration (171,000 ± 29,000/μL [mean ± standard error of the mean] versus 174,000 ± 29,000/μL). Furthermore, no spontaneous platelet aggregation or platelet aggregates were observed during the period of heparin exposure. The platelet count did decrease to 112,000 ± 17,000/μL with the inception of cardiopulmonary bypass and hemodilution but remained stable throughout the bypass period as well as during the postoperative periods (Fig 2). Similar results were ob-

Table 2. Hemodynamic Variables[a,b]

Variable	After Anesthesia	Before Heparin	After Heparin	Five Minutes After Bypass	Before Protamine	After Protamine
Mean arterial pressure (mm Hg)	94 ± 4	65 ± 3	64 ± 3	57 ± 4	69 ± 5	67 ± 3
Cardiac output[b] (L/min)	4.5 ± 0.5	5.1 ± 0.6	5.3 ± 0.5	. . .	5.4 ± 0.5	4.8 ± 0.4
Systemic vascular resistance[c] (dyne · s · cm^{-5})	1,392 ± 99	900 ± 102	906 ± 128	. . .	987 ± 148	1,087 ± 65

[a] Values are given as mean ± standard error. [b] The mean Neo-Synephrine dose was 73 ± 22 mg. [c] Excluding patient 1, who had no Swan-Ganz catheterization (tricuspid stenosis).

served in the patients undergoing vascular procedures (Table 3).

Platelet Factor 4 and β-Thromboglobulin

Mean plasma levels of PF4 increased from 30 ± 10 ng/mL after anesthesia induction and before heparinization to 591 ± 135 ng/mL immediately after heparinization. Platelet factor 4 levels decreased slightly with inception of cardiopulmonary bypass and hemodilution and continued to decrease after administration of protamine sulfate (Fig 3).

After induction of anesthesia, β-TG levels increased from 156 ± 45 to 235 ± 48 ng/mL after heparin administration, decreasing slightly to 199 ± 37 ng/mL five minutes after inception of cardiopulmonary bypass (Fig 4). Similar results for both PF_4 and β-TG were noted in patients undergoing vascular operations (Table 3).

Thromboxane B_2

Thromboxane B_2 concentrations increased from 441 ± 67 pg/mL after anesthesia to 942 ± 95 pg/mL after heparinization. These levels remained unchanged after inception of bypass (825 ± 106 ng/mL) (Fig 5). Thromboxane B_2 concentrations were highly variable from patient to patient, and were not assessed in patients undergoing vascular operations.

Fibrinopeptide A

Plasma levels of FPA decreased from 153 ± 70 ng/mL after induction of anesthesia to 27 ± 6 ng/mL with heparinization, remaining essentially unchanged during the bypass and postoperative periods (Fig 6). Similar results were obtained for patients undergoing vascular operations (Table 3).

Platelet Reactivity

All patients (cardiac and vascular) showed complete prevention of heparin-induced platelet aggregation after iloprost infusion just before heparin administration. No spontaneous platelet aggregation was observed during the period of heparin exposure. However, in the postoperative period, 7 of 11 patients still showed a strong in vitro aggregation response (≥20%) to heparin, indicating the persistence of the heparin-dependent platelet-aggregating factor (not shown).

Reactivity of platelets challenged by ADP was assessed to determine responsiveness throughout the perioperative period and to serve as a frame of reference for the

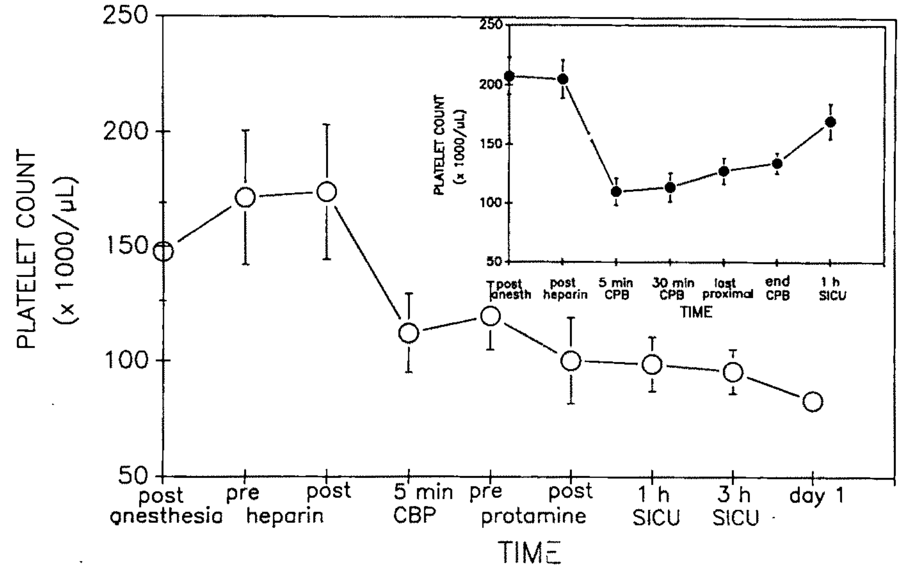


Fig 2. Mean platelet counts (± SEM) are plotted against time and expressed ×10^3/μL for patients with heparin-induced thrombocytopenia (○—○; n = 9) and for control subjects undergoing coronary revascularization (inset; ●—●; n = 21). (CPB = cardiopulmonary bypass; SICU = surgical intensive care unit.)

Table 3. Platelet Data From Patients Undergoing Vascular Procedures

	After Anesthesia/ Before Heparin		After Heparin	
Variable	Patient 11	Patient 12	Patient 11	Patient 12
Platelet count ($\times 10^3/\mu L$)	255	350	256	362
Platelet factor 4 (ng/mL)	19	23	200	315
β-Thromboglobulin (ng/mL)	56	76	96	107
Fibrinopeptide A (ng/mL)	40	. . .	6.4	. . .
Adenosine diphosphate aggregation (%)	56	71	15	1.25
Iloprost levels (pg/mL)	0	0	1,770	1,800

extent of aggregation observed after exposure of platelets to heparin alone. Platelet reactivity to threshold concentrations of ADP was inhibited (89% ± 4%) during iloprost infusion just before heparinization. Inhibition decreased to 53% ± 12% after heparin administration (Fig 7). Similar results were observed for patients undergoing vascular procedures (Table 3).

Iloprost Levels

Plasma concentrations of iloprost reached 1,758 ± 329 and 2,053 ± 576 pg/mL before and immediately after heparinization. Iloprost levels decreased to undetectable levels in the surgical intensive care unit period, reflecting the infusion protocol (Fig 8). Similar levels were observed in vascular surgical patients (Table 3).

Template Bleeding Time

Template bleeding times preoperatively and postoperatively were 7.9 ± 1.2 and 9.6 ± 1.3 minutes, respectively (Fig 9). The bleeding time of patient 1 was more than 20 minutes postoperatively and was assigned a value of 20 minutes for data collation.

Chest Tube Drainage

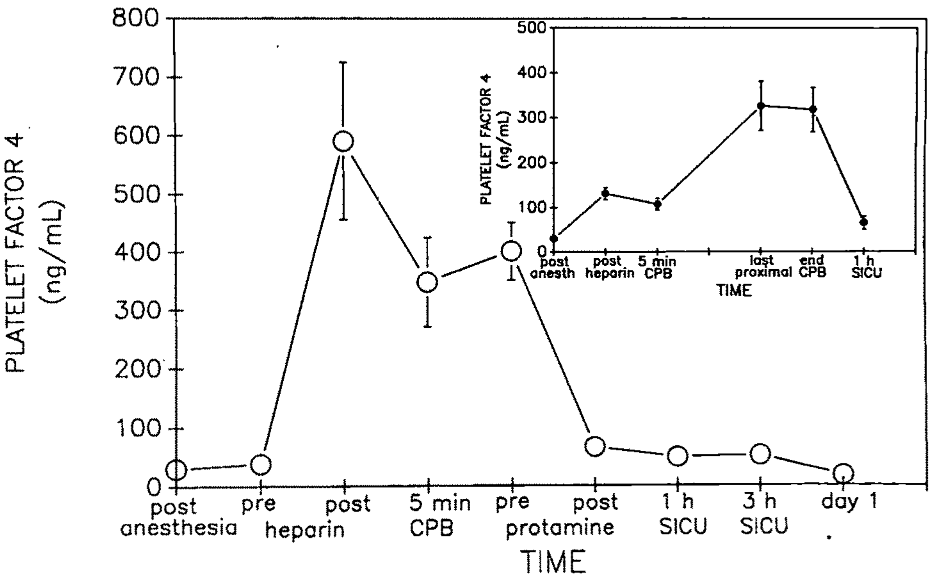
At six and 12 hours postoperatively, chest tube drainage was 356 ± 111 and 593 ± 185 mL, respectively (Fig 10). However, patient 7 appeared to bleed excessively (1,225 and 2,030 mL at six and 12 hours, respectively). Mean chest tube drainage excluding this patient was 247 ± 23 at six hours and 413 ± 48 at 12 hours.

Comment

The properties and mechanisms of heparin-platelet interactions remain incompletely characterized. A transient, mild thrombocytopenia after heparin administration is a nearly universal yet clinically insignificant occurrence [2]. This mild "platelet agglutination," however, must be distinguished from a severe form of thrombocytopenia associated with thrombosis and hemorrhage that has been termed heparin-induced or heparin-associated thrombocytopenia [3, 4].

Investigators first recognized that recurrent arterial emboli would occur despite adequate anticoagulation with heparin. At that time, Roberts and co-workers [20] suggested that a heparin/immune reaction may be responsible for the thrombotic proclivity in these unusual patients, but it was not until 1972 that a heparin-dependent platelet antibody was identified in patients with HIT [21]. Subsequently, this heparin-dependent platelet aggregating factor was shown to be in the immunoglobulin G serum fraction, to fix complement to both platelets and endothelial cells, and to induce platelet aggregation through a complement-dependent and, at least in certain patients, thromboxane-independent pathway [4, 22, 23].

Fig 3. Mean plasma platelet factor 4 levels (± SEM) plotted against time and expressed in nanograms per milliliter of plasma for patients with heparin-induced thrombocytopenia (○——○; n = 9) and for control subjects undergoing coronary revascularization (inset; ●——●; n = 21). (CPB = cardiopulmonary bypass; SICU = surgical intensive care unit.)



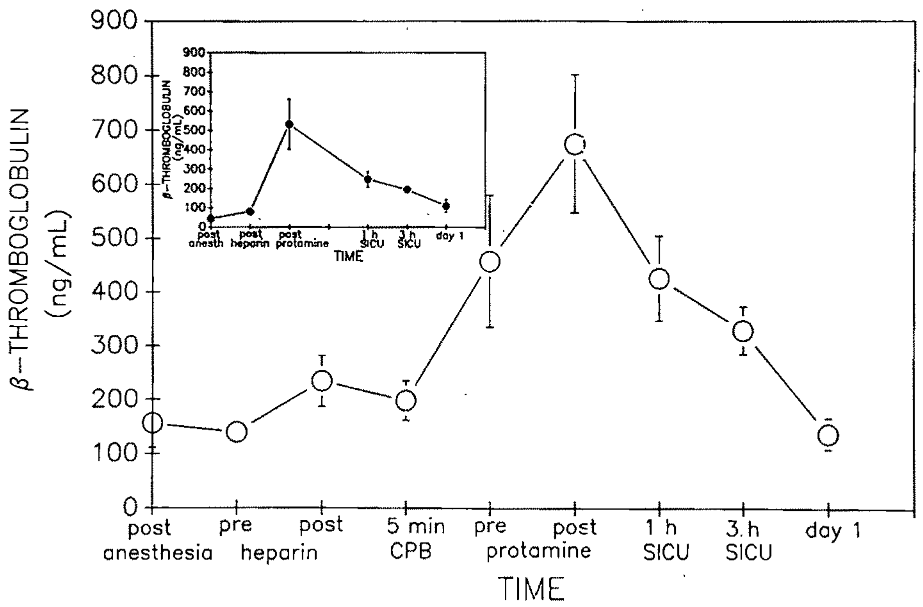


Fig 4. Mean plasma β-thromboglobulin levels (± SEM) plotted against time and expressed in nanograms per milliliter of plasma for patients with heparin-induced thrombocytopenia (○——○; n = 9) and for control subjects undergoing coronary revascularization (inset; ●——●; n= 7). (CPB = *cardiopulmonary bypass;* SICU = *surgical intensive care unit.)*

If patients receiving heparin experience HIT with thrombosis, the morbidity and mortality have been reported to be as high as 61% and 23%, respectively [3]. With careful surveillance and early detection, the morbidity can be reduced to 22.5% and the mortality to 12% [24]. Nevertheless, once the diagnosis of HIT is confirmed, cessation of all heparin, including heparin flushes and even heparin-bonded catheters, is strongly recommended. Some patients with HIT, however, may require cardiac or vascular procedures that are sometimes urgent. Heparin administration is favored for cardiac and vascular procedures for occlusive atherosclerotic disease. Thus, for these patients, reexposure to heparin is likely and other clinical strategies are necessary to prevent the sequelae of heparin-induced thrombosis or hemorrhage.

By waiting until the plasma activity of the heparin-induced platelet aggregating factor disappeared, Olinger and colleagues [25] were able to use a single dose of heparin successfully during cardiopulmonary bypass for 3 patients with previously documented HIT. This approach, however, is complicated by the variable length of time required for the plasma activity to decrease and thus would not be feasible for patients requiring urgent operation. For example, patient 11 in our series showed heparin-induced platelet aggregation and release 7 years after her last documented exposure to heparin. Furthermore, in another of our patients, we used the strategy of Olinger and colleagues [25] but platelet activation after heparin administration was clearly evident, as measured by formation of microaggregates and extensive release of PF_4 and β-TG (patient 2) [8]. Thus, we do not recommend merely delaying operation.

Other strategies have been used, including use of low-molecular-weight heparin fractions, heparinoids,

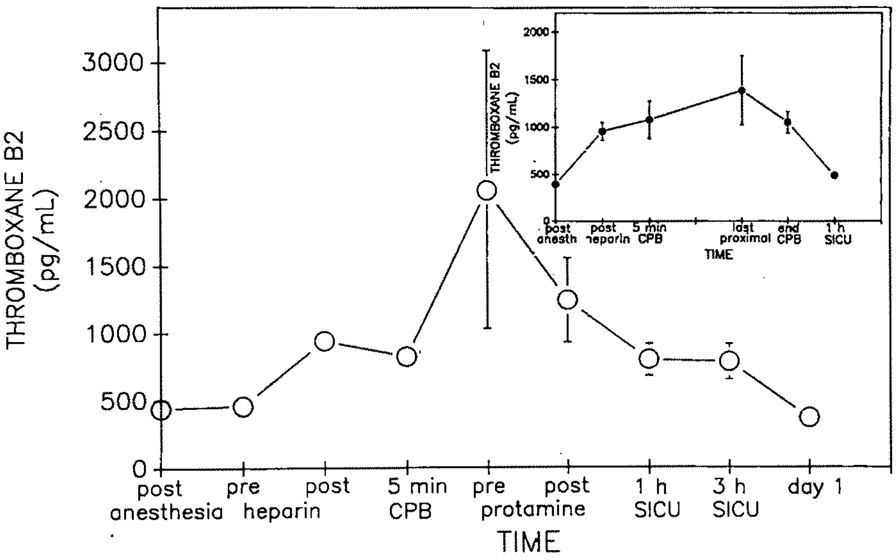
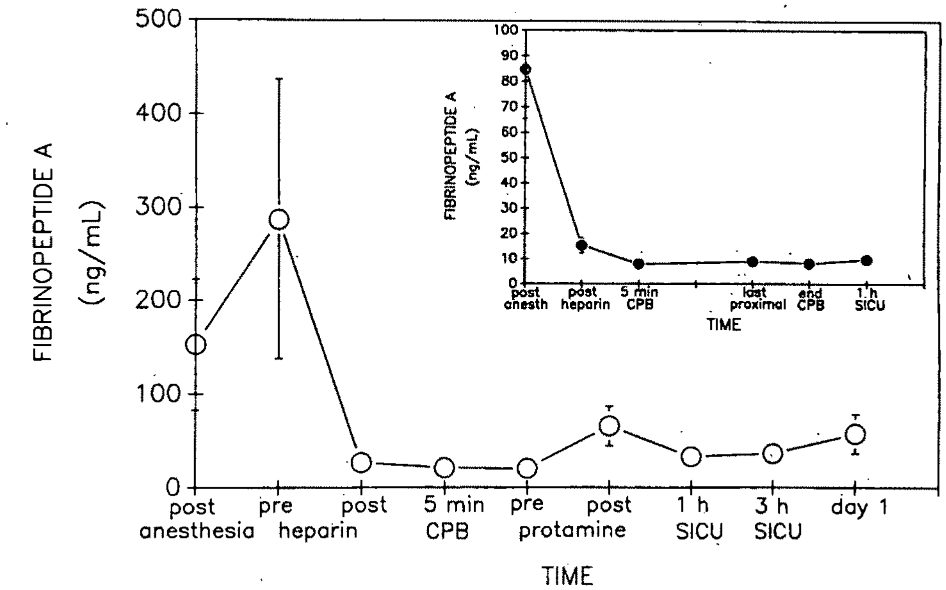


Fig 5. Mean plasma thromboxane B_2 levels (± SEM) plotted against time and expressed in picograms per milliliter of plasma for patients with heparin-induced thrombocytopenia (○——○; n = 9) and for control subjects undergoing coronary revascularization (inset; ●——●; n = 21). (CPB = *cardiopulmonary bypass;* SICU = *surgical intensive care unit.)*

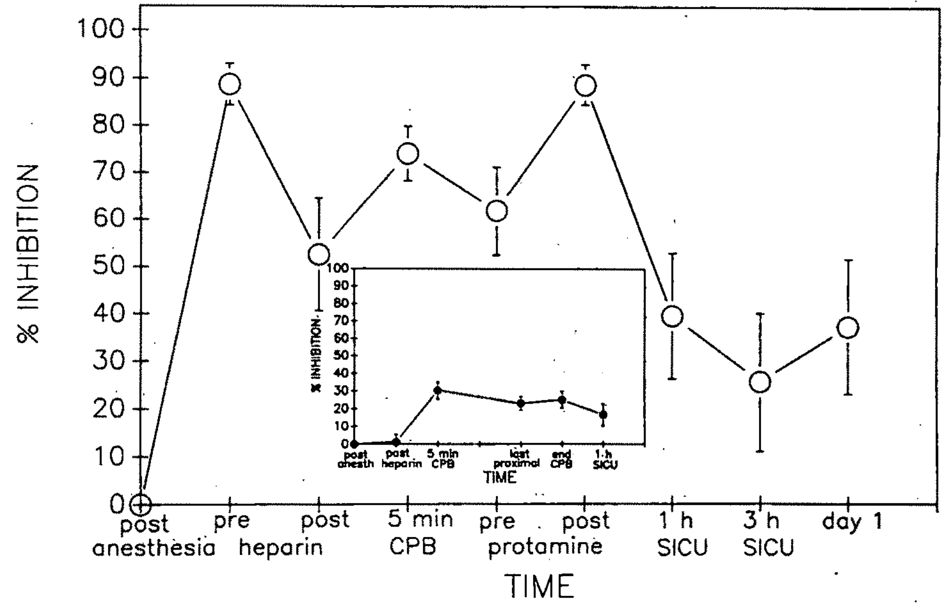
Fig 6. Mean plasma fibrinopeptide A levels (± SEM) plotted against time and expressed in nanograms per milliliter of plasma for patients with heparin-induced thrombocytopenia (O—O; n = 9) and for control subjects undergoing coronary revascularization (inset; ●—●; n = 21). (CPB = cardiopulmonary bypass; SICU = surgical intensive care unit.)



dextran, and Ancrod, a defibrinating agent, to avoid anticoagulation with heparin. Heparin is a "polydispersed" molecule. Theoretically, heparin fractions of low molecular weight that possess anticoagulant properties but that do not affect platelet function or trigger an antigenic response could be prepared. Unfortunately, use of low-molecular-weight heparin in patients with HIT has had unpredictable clinical results [26]. Heparinoids have been effective in vitro, but the lack of reversibility has made their clinical application problematic [27]. Finally, Ancrod has been used successfully at a number of centers but is also not easily reversed and may increase postoperative bleeding and transfusion requirements [28]. In contrast, with iloprost, heparin, which is still the anticoagulant of choice for extracorporeal circulation, can be safely administered. The noted decrease in systemic mean arterial pressure can be controlled with use of phenylephrine. On discontinuation of iloprost, phenylephrine requirements are no different from those for routine open heart operations.

Because most complications of heparin-induced platelet activation appear to be platelet mediated, pharmacological inhibition of platelet activation during a brief reexposure to heparin has considerable appeal [6, 7, 8, 29]. By irreversibly acetylating cyclooxygenase, aspirin prevents prostaglandin endoperoxide formation and thromboxane A_2 synthesis, a major pathway of platelet activation. Aspirin has been used to prevent heparin-induced platelet activation, but with variable efficacy [7, 29]. Furthermore, we have shown that heparin in certain patients can act as a strong platelet agonist and induce platelet activation with release of lysosomal granules through throm-

Fig 7. Platelet responsiveness (percentage of inhibition) to adenosine diphosphate plotted against time and expressed as a percentage of the initial percentage of aggregation (ie, percentage of light transmission to the adenosine diphosphate threshold dose obtained after induction of anesthesia) (± SEM) for patients with heparin-induced thrombocytopenia (O—O; n = 9) and for control subjects undergoing revascularization (inset; ●—●; n = 21). (CPB = cardiopulmonary bypass; SICU = surgical intensive care unit.)



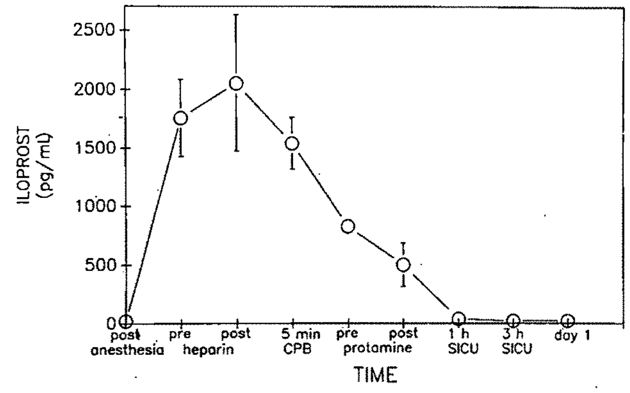


Fig 8. *Mean plasma levels of iloprost (± SEM) plotted against time and expressed as picograms per milliliter for patients with heparin-induced thrombocytopenia (n = 7; patients 8 and 9 excluded). (*CPB *= cardiopulmonary bypass;* SICU *= surgical intensive care unit.)*

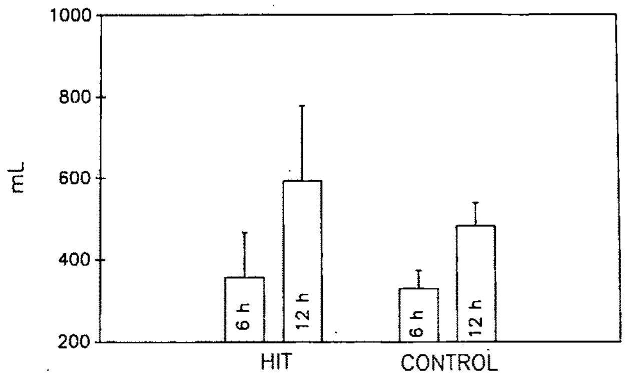


Fig 10. *Mean chest tube drainage amounts (± SEM) at six and 12 hours for patients with heparin-induced thrombocytopenia* (HIT) *(n = 9) and control subjects (n = 21).*

boxane-independent pathways [23]. Indeed, in 40% of the patients we have tested, aspirin failed to prevent heparin-induced platelet aggregation and release in vitro. Furthermore, because the effect of aspirin is irreversible, continued platelet dysfunction will persist into the postoperative period and may itself contribute to increased bleeding complications [30, 31]. Similarly, low-molecular-weight dextran has shown in vitro efficacy in concentrations that would be difficult to achieve clinically [32]. Thus, we do not recommend "conventional" antiplatelet therapy to manage these patients.

Iloprost (ZK 36374) is a stable analogue of prostacyclin, a potent endogenous inhibitor of platelet function. Iloprost effectively prevents platelet activation to all platelet agonists evaluated in our laboratory, including the strong agonist thrombin. Its mode of action is stimulation of adenyl cyclase and elevation of intracellular levels of cyclic adenosine monophosphate [33]. In nanomolar concentrations, a level that can be readily achieved clinically, iloprost in vitro prevented heparin-induced platelet aggregation and release in all patients evaluated (n = 11), including those in whom heparin appears to act through thromboxane-independent pathways [6, 7, 23]. In addition, because the inhibitory effects of iloprost do not outlast its presence in plasma, there is a rapid return of platelet function postoperatively as would be desired for hemostasis.

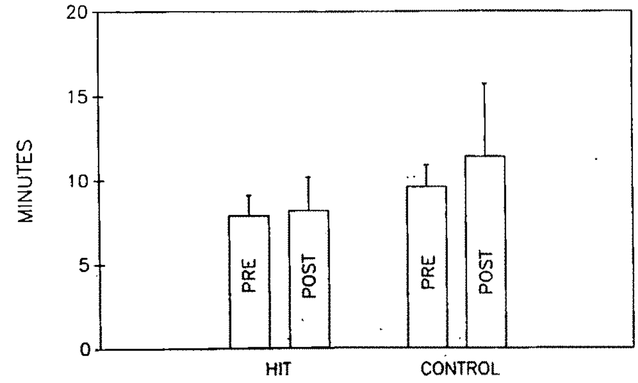


Fig 9. *Template bleeding time obtained immediately after anesthesia induction* (PRE) *and bleeding time obtained after arrival of patient in the surgical intensive care unit* (POST) *plotted for patients with heparin-induced thrombocytopenia* (HIT) *(n = 9) and control subjects (n = 21).*

Results in our patients show that an infusion of iloprost does prevent heparin-induced platelet activation. Indeed, in our group of patients, there was no change in circulating platelet count or formation of platelet aggregates after heparin administration. Similarly, FPA levels remained low, indicating absence of heparin-induced thrombosis. In addition, as assessed by postoperative bleeding times, the response to ADP, and postoperative chest tube drainage, platelet function was markedly preserved. Consistently, our patients, some of whom were valve recipients, possessed variables of postoperative platelet function remarkably comparable with those obtained from a historical control group from this institution, despite the presumed additive effects of synthetic surface-mediated and heparin-mediated platelet alterations.

Despite the marked preservation of platelet number and function, however, there was evidence of slight heparin-induced platelet activation in the iloprost group. For example, there was an increase in plasma levels of the platelet α-granule proteins PF_4 and β-TG after heparin administration. Although this degree of platelet activation appeared to be of no clinical significance, it does attest to the necessity of providing platelet inhibition during heparin administration. The appearance of platelet release products without platelet adhesion (ie, preserved platelet number) or platelet facilitated thrombosis (ie, low FPA levels throughout heparin exposure) suggests that prevention of adhesion of activated platelets to endothelial cells may actually serve as the primary mechanism of iloprost's efficacy [22].

No deaths were associated with heparin or iloprost administration in this clinical series even though several of our patients had severe cardiac dysfunction. Iloprost, like its parent compound, prostacyclin, is a vasodilator. In fact, vasodilatation has limited the clinical utility of prostacyclin in our experience. Fortunately, iloprost appears to be more manageable [33], and stable hemodynamic values were readily maintained with the aid of phenylephrine. Furthermore, no adverse side effects were identified that could be attributed to either hypotension or vasopressor administration. Thus, our experience to date has clearly demonstrated that iloprost can be administered safely even in this group of high-risk patients.

In summary, heparin-induced thrombocytopenia is associated with a morbidity and mortality as high as 61% and 23%, respectively. If the diagnosis of HIT is established, avoidance of reexposure to heparin is strongly recommended. For the select group of patients with HIT who require reexposure to heparin, some form of platelet inhibition is suggested. In our experience, iloprost provides this protection both safely and effectively, and is a valuable addition to the management armamentarium for these difficult patients.

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DISCUSSION

DR ANTHONY LEROY MOULTON (Omaha, NE): Dr Kappa, can you clarify how this group of high-risk patients was identified preoperatively? Was it only a history of problems with prior heparin exposure? Are there indications for preoperative testing for heparin sensitivity in patients undergoing cardiopulmonary bypass who do not have a history of problems with prior heparin exposure, or do you routinely test for heparin sensitivity?

DR ATTAR (Baltimore, MD): I have one question regarding the testing of the sensitivity of the patient's blood to heparin. You recommend that sensitivity to heparin be demonstrated by using ADP-induced aggregation in vitro preoperatively. In our laboratory, as well as in Dr O'Brien's in London and in Sweden, platelet aggregation does occur routinely if it is tested in vitro, whether one uses heparin from the intestines or lung heparin. How could this be prevented in the heparin-sensitive patients? Was it a quantitative difference or was it due to the specific type of heparin used?

DR RICHARD D. WEISEL (Toronto, Ont, Canada): I would like to compliment Dr Kappa on his excellent presentation and the group at the University of Pennsylvania for elucidating the mechanisms of platelet dysfunction after cardiac operations. In addition, they have evaluated alternative interventions in the clinical setting. Their study clearly documents that patients with HIT can be treated with iloprost.

Iloprost, however, does have some problems. It is a vasodilator, and in many patients vasoconstrictors were required to maintain blood pressure. If large concentrations of vasoconstrictors are required, inadequate perfusion of the kidneys, bowel, or brain could result. Vasoconstrictors may be associated with difficulties in occasional patients.

The mechanism for HIT was aggregation and degranulation of platelets in response to the heparin infusion. Dr Kappa, do you believe that other agents may also be beneficial in preventing the aggregation and degranulation response to heparin? We previously showed that an intravenous infusion of dipyridamole can prevent platelet degranulation, preserve platelet numbers, and reduce bleeding without producing systemic vasodilation. The group from the Netherlands has used aprotinin and have suggested that this agent protects platelets. Perhaps either dipyridamole or aprotinin may be beneficial in patients with HIT.

At the University of Toronto, we have used an alternative method to treat patients who cannot be given heparin. Under the direction of Dr Michael Glynn, our coagulation specialist, we have used the venom of the Malayan pit viper. The agent, Ancrod, is commercially available and has been used extensively in Europe to treat patients with thromboemboli. The agent is delivered intravenously preoperatively and depletes fibrinogen concentrations and therefore prevents thrombosis during cardiopulmonary bypass. However, both platelet function and hemostasis are maintained.

We used Ancrod in 6 patients with HIT, 10 patients with heparin resistance who could not be anticoagulated, and 6 patients with protamine-induced anaphylactic reactions. We also used the agent in 20 control patients. We had no morbidity or mortality in the patients who underwent cardiac operations with Ancrod rather than heparin. The Ancrod infusion increased postoperative bleeding and transfusion requirements. However, we had no other complications, and the patients recovered completely from their cardiac procedures. Therefore, we have one additional method of treating patients with either HIT, heparin resistance, or protamine-induced anaphylactic reactions.

DR KAPPA: Phenylephrine infusions were required. Iloprost is a vasodilator, although it seems to be less of a vasodilator than its parent compound, prostacyclin, or prostaglandin E_1. However, it has been used for platelet preservation during bypass in more than 100 patients now, and no complications with regard to renal failure, cerebrovascular accidents, or intestinal ischemia that we have noted can be directly related to iloprost.

Treating these patients is a very difficult problem. If nothing is done, arterial thrombosis to all four extremities can result. Indeed, such a case was reported during this meeting. Clearly, the best alternative would be to find another form of anticoagulation. Unfortunately, many alternatives, such as low-molecular-weight heparin, work in some patients but not in all. Other heparinoids have been effective at least in vitro in some models, but they lack specificity and cannot be reversed rapidly.

Aspirin did work in 40% of the patients that we tested in vitro. We demonstrated, however, that in certain patients heparin-induced platelet activation can proceed by thromboxane-independent mechanisms; this process would not be inhibited by aspirin. In addition, aspirin is a permanent platelet inhibitor and platelet dysfunction would still exist postoperatively.

We have evaluated dipyridamole in vitro. It probably has a mechanism of action in addition to its phosphodiesterase inhibition that has not yet been determined. We have not found it efficacious in vitro. Dipyridamole might enhance or be synergistic with iloprost, however, as they work by different mechanisms. Perhaps we could use lower doses of our drug and avoid some of the vasoactive side effects.

We have had no experience with Ancrod. I think that depleting plasma fibrinogen could result in a coagulopathy, which may explain the excessive postoperative bleeding.

With regard to indications for testing for HIT, many of these patients were referred to us after treatment elsewhere. Our indications for evaluating a patient for HIT are thrombocytopenia of less than 100,000 platelets per microliter during heparin administration, unexplained or ongoing thrombosis despite seemingly appropriate levels of heparin, or demonstration of increasing requirements for heparin. We do not recommend screening all patients who receive heparin. With regard to use of beef or porcine heparin, there is probably a difference in the incidence of HIT depending on the brand of heparin used. Once HIT has developed, however, we have found that cross-reactivity occurs between the two varieties of heparin. In testing for cardiac surgical patients, we used 3 units of heparin per milliliter, which corresponds roughly to the plasma levels obtained during open cardiac operations.

Indications for Esophagectomy in Nonmalignant Barrett's Esophagus: A 10-Year Experience

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Between 1978 and 1988, 88 patients were referred for the surgical treatment of nonmalignant Barrett's esophagus. Nineteen patients required esophageal resection. Male/female ratio was 13:6; age range was 13 to 84 years (mean age, 49.8 years; median age, 40 years). Preoperative studies demonstrated strictures in 11 patients and ulcers in 7. Penetrating Barrett's ulcer resistant to treatment was the indication for resection in 5 patients. Ulcers penetrated to the pericardium (1 patient), pulmonary vein (1), lung (1), and mediastinum (2). Other indications for resection included undilatable strictures (2), previous operations (4), high-grade dysplasia (3), parietal cells lining the esophagus (1), patient's refusal of long-term surveillance (2), and the inability to exclude adenocarcinoma preoperatively (2). Reconstruction was achieved by colon interposition (15) or esophagogastrostomy (4), with one postoperative death. Mean follow-up was 41 months and was 100% complete. Of the 18 patients, 3 have occasional regurgitation but none have any dysphagia or weight loss. Esophageal resection is indicated in a select group of patients with Barrett's esophagus. Absolute indications include a deep penetrating ulcer confirmed intraoperatively, high-grade dysplasia, strong suspicion of cancer, and multiple previous operations. Relative indications include strictures not responding to dilation and young patients refusing long-term surveillance.

(Ann Thorac Surg 1990;49:724–7)

Since its description by Norman Barrett in 1950 [1], the columnar-lined esophagus has been a focus of controversy. Confusion shrouds its definition, pathogenesis, therapy, and follow-up. It is now widely accepted that Barrett's esophagus is associated with long-standing severe gastroesophageal reflux. Resurfacing of ulcerative esophagitis apparently occurs by columnar cells. The origin of the glandular epithelium remains unclear. The therapy of Barrett's esophagus and its complications is still debated among gastroenterologists and surgeons. This report outlines our indications for esophagectomy in patients with benign Barrett's esophagus.

Material and Methods

Between 1978 and 1988, 88 patients were referred for the evaluation and treatment of nonmalignant Barrett's esophagus. Nineteen patients underwent esophagectomy and are the subject of this report. We diagnose Barrett's esophagus when at least 3 cm of the distal tubular esophagus is lined with columnar epithelium, regardless of the presence or absence of a hiatal hernia or when a biopsy of the distal tubular esophagus retrieves epithelium with intestinal metaplasia [2]. All nineteen patients met this definition. Patient records were carefully reviewed to determine the principal indication for esophagectomy in each case. When more than one indication was found, the principal indication was determined by the major preoperative working diagnosis. Follow-up is complete in all patients either to time of death or December 1988.

Results

There were 13 male and six female patients ranging in age from 13 to 84 years (mean age, 48.3 years; median age, 40 years). All patients were white. Eight patients had previous esophageal operations, one of whom had partial esophagectomy with intrathoracic esophagogastrostomy. Seven patients had previous antireflux repairs, including 1 who had three previous attempts.

Heartburn was present in 17 patients and varied in duration between 1 to 20 years (mean duration, 10 years). Dysphagia was present in 17 patients and regurgitation in 12. Three patients had severe atypical pain, all of whom had deep penetrating Barrett's ulcers: 1 had upper back pain misdiagnosed as thoracic spondylosis, 1 had severe epigastric pain, and the third patient had severe midchest pain requiring narcotics for relief.

Upper gastrointestinal series and esophagoscopy were performed on all patients. Strictures were noted radiographically and endoscopically in 14 patients. Ten patients had strictures located in the midesophagus. Ulceration in the columnar epithelium was noted in 10 patients at endoscopy, and ulcers were the only finding both radiographically and endoscopically in 4. The length of the metaplastic epithelium was measured endoscopically and ranged from 3 to 12 cm (mean length, 6 cm). All cases were biopsy-proven. Intestinal metaplasia was present in

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Table 1. Indications for Esophagectomy

Barrett's ulcer	5
Stricture	2
High-grade dysplasia	3
Rule out cancer	2
Parietal cell lining	1
Young patient	2
Multiple previous esophageal operations	4

5 patients, fundic epithelium was seen in 3, and junctional type in 7 patients. A combination of junctional type and intestinal type was seen in 2 patients, and all three types were seen in another 2. All biopsy specimens were reviewed for evidence of dysplasia, which was graded using the criteria established for inflammatory bowel disease [3]. High-grade dysplasia was noted in 3 patients.

Esophageal manometry was performed on 12 patients. The lower esophageal sphincter was atonic in 2 patients, whereas 10 had a hypotensive lower esophageal sphincter (mean lower esophageal sphincter pressure, 6 mm Hg). Twenty-four-hour pH monitoring was performed in 14 patients; the results were abnormal in all 14. Indications for esophagectomy in the entire group are shown in Table 1.

Penetrating Barrett's ulcers resistant to treatment were the principal indication for resection in 5 patients. Ulcers penetrated to the pericardium in 1 patient, pulmonary vein in 1, and lung tissue in 1. Penetration to unspecified sites in the mediastinum occurred in 2 patients. Severe atypical pain was present in 3 patients and hematemesis was the presenting feature in 1. One patient with an ulcer that penetrated to the inferior pulmonary vein had recurrent ulcerative esophagitis after a Nissen fundoplication done elsewhere.

Other indications for resection included strictures not responding to dilation (2 patients), multiple previous operations (4), and high-grade dysplasia (3). Two patients with high-grade dysplasia had intestinal-type epithelium. Carcinoma was not found in the resected specimen in any of the 3 patients with high-grade dysplasia. One resected patient with parietal cell lining of the esophagus had recurrent strictures and ulcerations after a previous Nissen fundoplication. Two other young patients, 1 of whom had intestinal-type epithelium without dysplasia on preoperative biopsies, opted for esophagectomy over long-term endoscopic surveillance. Adenocarcinoma could not be definitively excluded preoperatively in 2 other patients. Although the resected specimen in both patients showed large penetrating ulcers with chronic mediastinitis, the preoperative diagnosis favored adenocarcinoma.

The colon was the viscus of choice for reconstruction and was employed in 15 patients. The stomach was used in 4 patients. There was one hospital death (5.2%) occurring in an 84-year-old man with Barrett's ulcer that penetrated into the mediastinum. Follow-up is 100% complete until December 1988. Seventeen patients have neither dysphagia, heartburn, nor weight loss. Three patients have occasional episodes of regurgitation. One patient died 5 years postoperatively of unrelated causes and was asymptomatic at the time of death.

Comment

This report confirms the widely accepted opinion that Barrett's esophagus is associated with, and possibly complicates, long-standing gastroesophageal reflux. Seventeen patients had heartburn and regurgitation for a mean duration of 10 years. Twelve patients evaluated by both manometry and 24-hour pH monitoring had severe gastroesophageal reflux and associated profound incompetence of the cardia. This is in accordance with previously reported manometric and 24-hour pH data in patients with Barrett's esophagus [4]. The sex distribution is similar to that seen in our overall population with benign Barrett's esophagus, in which 60% are male. This is in contrast to patients with malignant Barrett's esophagus, among whom the male to female ratio is greater than 9:1.

Penetrating Barrett's ulcers were the most common indication for resection in our patients. An ulcer that penetrated through the esophageal wall into the mediastinum or other intrathoracic structures was present in 7 patients, and was the principal indication for esophagectomy in 5. It is of interest that 4 of those 5 patients were seen with manifestations that predicted the likelihood of penetration through the esophageal wall; 3 patients had severe atypical pain, 1 of whom required narcotics for relief, and another was being evaluated for thoracic vertebral spondylosis. A fourth patient had two episodes of hematemesis requiring transfusion. In 1 patient the ulcer was found intraoperatively to have penetrated to the inferior pulmonary vein.

The management of penetrating Barrett's ulcer is a difficult and controversial issue. Conservative medical management will usually suffice for the treatment of superficial ulcerations seen in the majority of patients with Barrett's esophagus. Penetrating ulcers either fail to respond or promptly recur with cessation of therapy. Pearson and associates [5] employed a Collis-Belsey antireflux repair in a group of 11 patients with complete healing of the ulcer in 8. An alternative approach is primary esophagectomy, as some of the ulcers are found to penetrate beyond the anatomical esophagus at the time of operation. Generally, we prepare our patients with Barrett's ulcers for the possibility of esophagectomy. The final decision regarding resection versus an antireflux repair is made intraoperatively, according to the integrity of the esophageal wall. When the ulcer does not penetrate through the esophageal wall an antireflux repair is appropriate and sufficient [5, 6]. Esophagectomy is reserved for those patients in whom an ulcer penetrates through the full thickness of the esophageal wall.

Two patients in this series required resection for strictures not responsive to dilation. They represent 8% of a series of 24 patients with Barrett's strictures previously reported [7], treated primarily with dilation and antireflux repair. Three patients underwent esophagectomy for high-grade dysplasia in Barrett's epithelium, 2 of whom had intestinal metaplasia. Carcinoma was not found in any of the resected specimens. We have previously reported on 4 patients who underwent esophagectomy for high-grade dysplasia without preoperative evidence of carcinoma [2, 8]. Three of those patients had invasive carcinoma in the resected specimen. We regard high-grade dysplasia as a serious indicator for malignant transformation in Barrett's esophagus and consider it an indication for esophagectomy if patients are suitable surgical candidates.

The precise definition of dysplasia is still debated in the literature. Some define dysplasia as an unequivocal neoplastic alteration, ranging from the large hyperchromatic nuclei confined to the basal parts of the cells in low-grade dysplasia to the total loss of nuclear polarity seen in high-grade dysplasia [9]. Differentiation between high-grade dysplasia and carcinoma in situ can be exceedingly difficult, even for experienced cytopathologists [10], and some include carcinoma in situ within the spectrum of high-grade dysplasia [9].

Esophagectomy was performed on 4 patients with multiple previous operations on the esophagus. Three patients had one to three previous attempts at antireflux repair, and 1 had a previous partial esophagectomy for Barrett's stricture with intrathoracic esophagogastrostomy. Two years later he was seen with dysphagia and severe weight loss. Endoscopy revealed recurrent Barrett's esophagus with stricture and ulceration. An esophagectomy with colon interposition was performed, recovery was uneventful, and he remains well to date. This case lends some support to the acquired theory of Barrett's esophagus. Intrathoracic esophagogastrostomy is frequently fraught with the hazard of continued reflux, and we generally do not employ this procedure for reconstruction of the esophagus after esophagectomy for benign disease.

One patient was seen 2 years after a Nissen antireflux repair performed elsewhere with recurrent ulcerations and stricture. Endoscopy and upper gastrointestinal series demonstrated an anatomically intact fundoplication, and manometry revealed a sphincter pressure of 10 mm Hg. Biopsy showed Barrett's esophagus of the fundic type. Recurrence of her stricture was attributed to pooling of acid secreted by the parietal cells lining the esophagus above a total fundoplication. This diagnosis is speculative because 24-hour pH monitoring cannot differentiate between a functional failure of the repair and acid production by parietal cells lining the esophagus. The ability of heterotopic or metaplastic parietal cells to secrete acid has been well documented, but its clinical significance is unclear [11–13]. This is certainly a small group of patients with Barrett's esophagus, whose therapy should merit further study. The presence of parietal cells lining the esophagus cannot be considered an indication for primary resection at this time. A partial fundoplication probably prevents acid pooling above the repair, and thus the possibility of persistent or recurrent esophagitis.

Finally, esophagectomy was performed on 2 young patients, aged 31 and 36 years, both of whom opted for esophagectomy over long-term endoscopic surveillance of the abnormal epithelium. One of the 2 had intestinal metaplasia with high-grade dysplastic changes in his resected specimen, and the other had extensive parietal cell lining of the esophagus. This indication for resection is a relative one, especially in light of recent information suggesting that the risk of developing cancer, although real, is less than previously thought [14].

Esophageal resection is indicated in a select group of patients with Barrett's esophagus. Absolute indications include a deep, penetrating ulcer confirmed intraoperatively, high-grade dysplasia, strong suspicion of cancer, and multiple previous operations on the esophagus. Relative indications include strictures not responding to dilation and young patients refusing long-term surveillance. The operative mortality in this series was gratifyingly low, encouraging us to consider this therapeutic option in some patients with the columnar-lined esophagus.

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DISCUSSION

DR ROBERT J. GINSBERG (Toronto, Ont, Canada): This report once again presents another problem area in the management of Barrett's esophagus: Who should have an esophagectomy? From the extreme of everybody diagnosed with Barrett's esophagus to only those with carcinoma of the esophagus, somewhere in between should be a proper rational approach to esophagectomy for this disease.

With regard to Barrett's ulcers, Dr Pearson in our own group (Ann Thorac Surg 1987;43:241–4) reported last year successful healing of all Barrett's ulcers after an antireflux operation. I can assure you that although many of those patients had penetrating ulcers, no esophagectomies were performed. Pylorectomy and duodenectomy were never recommended for penetrating ulcers of the duodenum, and certainly a good antireflux procedure will ensure healing of most, if not all, Barrett's ulcers. We would therefore not consider penetrating ulcers as an absolute indication for esophagectomy.

The group represented by Dr Altorki has used a colon interposition, and in their report they do not indicate how this colon interposition was done. Certainly many of the squamocolumnar junctions are above the carina. Dr Altorki, did you ensure that all columnar-lined epithelium was removed? Were these colon interpositions done by left thoracotomy and interposition, or were these total interpositions with anastomosis in the neck?

Other than carcinoma, our indications for esophagectomy include severe dysplasia on repeated biopsies, and certainly carcinoma in situ. Concerning the indication of esophagectomy for refusal of long-term surveillance, I certainly would try to convince those people that an esophagoscopy once a year or once every other year certainly would be preferable to esophagectomy. I am not sure this is a valid indication.

DR HOWARD S. BROWN (Atlanta, GA): Dr Altorki, how do you define severe dysplasia? Is this strictly a pathological diagnosis? Is the definition a combination of appearance plus pathology? Also, would you do the same procedure for minimal dysplasia or only for severe dysplasia?

DR STANLEY C. FELL (Chappaqua, NY): Like Dr Ginsberg, I am interested in where the esophagocolic anastomosis was placed in the chest. There is at least one well-documented case in the literature in which the Barrett's esophagus also was excised, an isoperistaltic colon interposition was placed, and a stricture subsequently developed at the cologastric anastomosis, effectively preventing reflux, and yet malignancy in the residual intrathoracic esophagus, a Barrett's carcinoma, developed.

DR ALTORKI: In response to Dr Ginsberg, we certainly agree that a Barrett ulcer diagnosed preoperatively is not an absolute indication for resection. We very heavily emphasize that that decision is made in the operating room at the time of exploration. Those patients are generally prepared for the possibility of esophagectomy with an interposition with colon or stomach. If, however, the ulcer has totally penetrated outside the boundaries of the anatomical esophagus, we feel that at that point an esophagectomy would better serve the patient.

In regard to the conduit that we use, you notice that we did use the colon. The mean length of the Barrett's esophagus in our series was about 6 cm, some of them extending above the arch. Those were reconstructed in the neck.

We do insist on a squamous mucosa in the proximal esophagus at the time of making the anastomosis, and we do get frozen sections of that. Out of the 15 colon segments, three were in the neck and 12 were in the mediastinum; out of the four gastric ones, three were in the neck.

With regard to high-grade dysplasia, we acknowledge the controversy involved, which extends even to pathologists. Some pathologists find it very difficult to differentiate high-grade dysplasia from carcinoma in situ. We insist that two different pathologists review the slides before making an absolute diagnosis of high-grade dysplasia, and before committing the patient to an esophagectomy. Whether they call it high-grade dysplasia or carcinoma in situ, I think from a clinical and a practical standpoint the important thing is that it is not invasive carcinoma.

I think the point about young patients refusing long-term surveillance is certainly well made, and we do advocate as much as possible that those patients undergo serial esophagoscopy with surveillance rather than esophagectomy. Our patients in this study were advised of that option but both opted for resection instead.

Role of Brachytherapy in the Management of Pulmonary and Mediastinal Malignancies

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Brachytherapy, the permanent or temporary implantation of radioactive sources, has been performed in limited numbers of patients with lung cancer over the last 50 years. Because of renewed interest in this modality, we reviewed our experience with 103 patients treated over a 7-year period. The mean age of this group was 55.5 years (range, 1 to 84 years). Primary lung cancer accounted for 82 patients (79.6%); metastatic lesions to the lung, 13 (12.6%); and mediastinal malignancies, 8 (7.8%). Indications for brachytherapy included mediastinal and chest wall invasion in 42 patients (40.8%), unresectable tumors and mediastinal adenopathy in 30 (29.1%), medical contraindications to extensive pulmonary resection in 20 (19.4%), and irradiation of excised lymph node beds in 11 (10.7%). Seeds labeled with radioactive iodine 125 alone were used in 65 patients (63.1%), afterloading catheters containing iridium 192 sources in 25 (24.3%), and both in 13 (12.6%). There were no operative deaths. With a mean follow-up of 18.6 months, the mean and median survivals for the entire group were 17.3 and 14.0 months, respectively. The 1-year, 2-year, and 3-year survivals for the entire group were 67.9%, 38.7%, and 27.8%, respectively. In summary, brachytherapy offers a useful surgical approach in patients in whom unresectable pulmonary or mediastinal malignancies are found at the time of thoracotomy or in patients previously treated with other modalities for whom limited therapeutic alternatives exist.

(Ann Thorac Surg 1990;49:728–33)

The goals of cancer treatment include eradication of all malignant cells when possible or destruction of the largest volume of malignant tissue when palliation is sought. By the use of proper staging procedures, most chest malignancies are operated on with curative intent. Despite the high predictive accuracies of these staging procedures, a small number of thoracotomies will expose incurable disease. In these instances, usually only two options exist: debulk or excise the tumor or terminate the operation.

Brachytherapy (BT) (implantation of seeds labeled with iodine 125 [^{125}I] or placement of afterloading catheters with iridium 192 [^{192}Ir] sources or both) offers a reasonable third alternative. To document the value of this mode of therapy, the records of all patients with lung or mediastinal malignancies treated by BT at Henry Ford Hospital were examined.

Material and Methods

Between November 1, 1980, and December 27, 1987, 103 patients with chest malignancies were treated by BT. The mean age of this group was 55.5 ± 16.5 years with a range of 1 year to 84 years. The male to female and white to black ratios were both 2.4:1. Primary lung cancer accounted for 82 patients (79.6%); metastatic lesions to the lung, 13 (12.6%); and mediastinal malignancies, 8 (7.8%). Cell types for primary lung carcinoma included squamous cell in 42 patients (51.3% of this subgroup), adenocarcinoma in 30 (36.6%), undifferentiated in 4 (4.9%), small cell in 2 (2.4%), carcinoid in 2 (2.4%), bronchoalveolar in 1 (1.2%), and large cell in 1 (1.2%). In this subgroup, 6 patients (7.3%) were in stage I, 3 (3.7%) were in stage II, and 73 (89.0%) were in stage IIIA or IIIB. Metastatic lesions to the lung consisted of osteogenic sarcoma in 3 patients (23.0%), synovial cell sarcoma and hemangiopericytoma in 2 (15.4%) each, and chondrosarcoma, breast, colon, testicular, acinar parotid, and thyroid carcinoma in 1 (7.7%) each. Malignant mediastinal masses included thymoma in 2 patients (25.0%) and Hodgkin's disease, thyroid, esophagus, neuroblastoma, thymic carcinoid, and mesothelioma in 1 (12.5%) each.

Early in this series, the decision to use BT was made during thoracotomy when an unexpected unresectable situation was encountered. Radiotherapists trained in BT administration were called to the operating room for implantation of sterile ^{125}I seeds into lung tumors. Gradually this use expanded to unresectable pulmonary metastases and malignant mediastinal masses. Because the initial analysis of results was favorable [1, 2], some patients were offered BT as an adjunct to irradiation or chemotherapy (or both). Patients with small cell carcinoma were not considered for BT unless all treatment options had been exhausted. Indications for BT included

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mediastinal and chest wall invasion in 42 patients (40.8%), unresectable tumors and mediastinal adenopathy in 30 (29.1%), medical contraindications to extensive pulmonary resection in 20 (19.4%), and irradiation of excised malignant lymph node beds in 11 (10.7%). Brachytherapy was planned in 52 patients (50.5%).

Most primary lung carcinomas were approached through a standard posterolateral thoracotomy; bilateral lung lesions and most mediastinal masses were exposed by a median sternotomy. Excision or debulking of the main tumor mass was performed when possible; any remaining malignant tissue was implanted with ^{125}I-labeled seeds (3M Health Care Group, St. Paul, MN) (approximately 0.5 mCi per seed) using the Mick applicator (Mick Radio-Nuclear Instruments, Bronx, NY). After caliper measurement of tumor volume, the modified Memorial Sloan-Kettering nomogram [3] was used to calculate the total activity and the number of seeds needed. Seventeen-gauge needles, 10 or 20 cm long, were used to space the seeds uniformly throughout the tumor. In general, malignancies greater than 6 cm in diameter were not considered for BT.

Two to three days after operation, orthogonal simulation films for dosimetric calculations were made in the Radiation Oncology Department. Isodose curves were generated for verification of dose distribution and calculation of a peripheral dose of the implanted volume.

Temporary ^{192}Ir sources were implanted through afterloading catheters (Best Industries, Inc, Springfield, VA). At thoracotomy, a 17-gauge needle was inserted through the chest wall through a low interspace. A sterile, closed-ended plastic catheter was threaded through the needle into the thoracic cavity. After needle withdrawal, the catheter was placed in the area to be irradiated. This procedure was repeated at 1-cm intervals until the desired number of catheters was placed. An absorbable suture material, usually chromic catgut, secured the catheters to the underlying pleura. Careful measurements of the exact area of irradiation were recorded. A removable steel cable was left in the catheter for support. Two to three days after operation, simulation films and dosimetric calculations were made.

Postoperative care was similar to that of other thoracic surgical patients with one exception. By Nuclear Regulatory Commission requirements, patients with catheters containing ^{192}Ir were isolated in designated private rooms with portable lead-shielding while the active seeds were in place. These catheters usually were loaded with seeds labeled with ^{192}Ir during the first two to three postoperative days and left in place 72 hours to give approximately 3,000 cGy to the resected bed. In general, chest tubes were not removed until the radioactive sources were withdrawn to prevent catheter dislodgment. Patients who had not received external-beam radiation therapy were treated after discharge with approximately 5,000 cGy over a 5-week period. Chemotherapy was not routinely employed postoperatively except in patients with small cell carcinoma. It was reserved for those who demonstrated progression of disease after BT and external-beam irradiation. A minimum of 1 year was allowed for follow-up of each surviving patient in this series.

Table 1. Complications of Brachytherapy[a]

Complication	No. of Patients
Prolonged air leak	5
Atelectasis	5
Prolonged fever	5
Respiratory failure[b]	4
Arrhythmia	3
Pneumonitis[b]	3
Pleural effusion[b]	3
Minor hemoptysis	2
Retained afterloading catheter	1
Ileus	1
Wound infection	1
Urine retention	1
Bronchospasm	1
Postoperative bleeding	1
Innominate vein thrombosis	1
Esophageal stricture[b]	1
Thrombocytosis[b]	1

[a] Seventy-two patients had no complications, and there were no operative deaths. [b] Some patients had more than one complication.

Results

Preoperative external-beam radiation therapy and chemotherapy were used in 23 (22.3%) and 22 (21.4%) patients, respectively. Prior thoracotomy had been performed in 14 (13.6%). The ^{125}I-labeled seeds were implanted in 65 patients alone (63.1%), the catheters with ^{192}Ir sources were loaded in 25 (24.3%), and both were used in 13 (12.6%). Lobectomy was performed in 13 patients (12.6%), pneumonectomy in 7 (6.8%), wedge resection in 13 (12.6%), and debulking of tumor in 17 (16.5%); no resection was done in 53 (51.5%). A standard lymph node dissection of potentially involved beds was carried out in 38 patients (36.9%). When used, an average of 42.7 seeds labeled with ^{125}I were implanted (range, 5 to 103), yielding an average of 23.2 mCi per patient; a mean of 3.4 catheters with ^{192}Ir were employed per patient (range, 2 to 7), producing a mean dose of 2,704 cGy. Postoperative radiation therapy was employed in 54 patients (52.4%) with a mean dose of 4,801 cGy (range, 650 to 6,800 cGy). Chemotherapy was used in 12 (11.6%), combined chemotherapy and radiation therapy in 19 (18.4%), and neither in 18 (17.5%).

Postoperative complications occurred in 30.1% of patients (Table 1). Most were minor and required no extensive treatment. Major complications included prolonged air leak, clinically significant atelectasis, and prolonged fever in 5 patients (4.8%) each, transient respiratory failure in 4 (3.9%), pneumonitis in 3 (2.9%), and retained afterloading catheter, postoperative bleeding, innominate vein thrombosis, and esophageal stricture in 1 (1.0%) each. There were no operative deaths.

Five patients (4.8%) were lost to follow-up; drop-out times occurred at 4, 10, 10, 22, and 52 months. With an average follow-up of 18.6 months, the mean and median

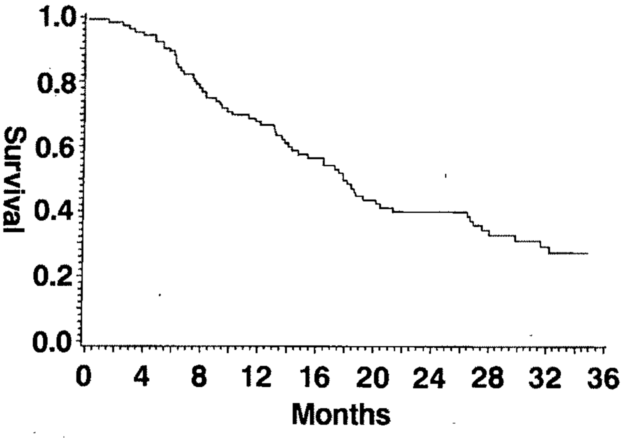

Fig 1. Kaplan-Meier survival curve for all patients from the date of brachytherapy.

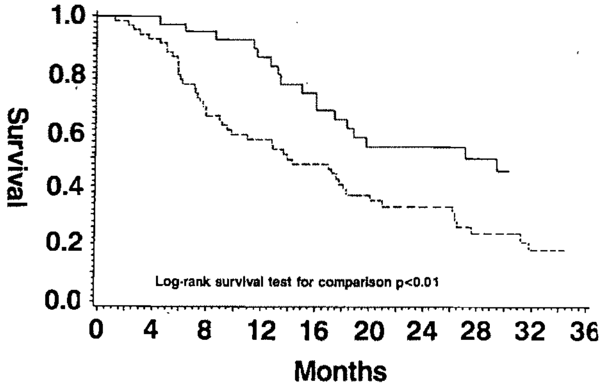

Fig 3. Kaplan-Meier survival curves from the date of brachytherapy comparing lymph node resection (solid line) *with no resection* (broken line).

survival times for all patients were 17.3 and 14.0 months, respectively. Figure 1 shows the Kaplan-Meier survival curve for the total patient population. Survival curves for subgroups with tumor resection or debulking versus implantation only and lymph node bed excision versus no resection are presented in Figures 2 and 3, respectively. The curves within these subgroups are significantly different with p values of less than 0.01 by log-rank analysis. No significant differences in survival were seen between the various cell types of malignancy, the type of malignancy (primary lung carcinoma versus metastatic lesions to the lung or primary mediastinal malignancies), the stage of primary lung cancer, or the type of BT used. Table 2 presents survival figures for various subgroups of patients with chest malignancies treated with BT.

Of those surviving 12 months, 15.2% had local recurrence, 22.0% had metastatic disease only, 11.9% had local and metastatic disease, and 50.9% had no measurable tumor. At 24 months, local disease was present in 12.5% of survivors, metastatic problems in 15.6%, both local and metastatic disease in 15.6%, and none in 56.3%. At 12 and 24 months, 72.9% and 71.9%, respectively, had no evidence of local disease. At death (n = 77), local disease only was present in 29 patients (37.7%), metastatic disease only in 18 (23.4%), both in 20 (26.0%), and none in 10 (13.0%). Of those alive and available for follow-up (n = 21), local disease was present in 2 patients (9.5%), metastatic disease in 2 (9.5%), both in 1 (4.8%), and none in 16 (76.2%). At the time of this report, 10 patients had survived longer than 36 months, 6 lived past 48 months, and 1 lived past 60 months. Of the 5 patients lost to follow-up, no active disease was present at their last examination.

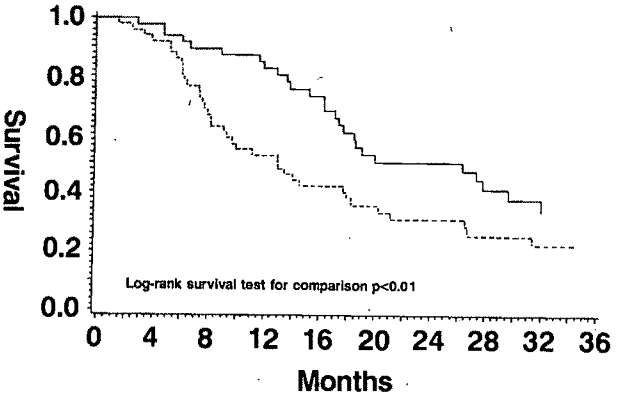

Fig 2. Kaplan-Meier survival curves from the date of brachytherapy comparing tumor resection or debulking (solid line) *with implantation of iodine 125–tagged seeds only* (broken line).

Comment

Sporadic attempts to treat malignant lesions with interstitial implantation of radioactive substances occurred in the early part of the 20th century [4]. In 1933, Graham and Singer successfully used radioactive radon seeds in the pneumonectomy stump of a patient operated on for lung cancer [5]. Little interest in this mode of cancer treatment existed until the introduction of ^{125}I in 1965. This isotope emits low-energy gamma rays with an activity field ap-

Table 2. Survival Estimates for Various Subgroups of Patients with Chest Malignancies Treated With Brachytherapy[a]

	Survival (%)		
Variable	12 mo	24 mo	36 mo
All patients	67.9	38.7	27.8
Type of malignancy			
Primary lung carcinoma	67.3	38.7	25.8
Metastatic lesions to lung	76.9	51.3	32.0
Mediastinal malignancies	62.5	37.5	37.5
Cell types			
Adenocarcinoma	75.3	43.7	35.0
Squamous carcinoma	60.8	33.6	14.9
Type of brachytherapy			
Iodine 125 alone	59.3	37.4	24.2
Iridium 192 alone	87.1	43.5	36.2
Both	69.2	46.1	30.8

[a] Log-rank analysis showed no significant differences between subgroups.

proximately 1 cm around the source. In aggregate, ^{125}I-labeled seeds can deliver a very high dose of radiation within a delimited volume of tumor. These qualities plus an abbreviated half-life of 60 days make it an ideal source for short-term interstitial irradiation. Iridium 192 has a longer half-life (74 days) and a higher energy gamma emission. For these reasons, afterloading catheters must be used to allow removal of the ^{192}Ir-tagged sources after delivery of the desired local radiation dose. Because the radius of activity is small (1 cm) with both sources, this form of BT can be used in areas previously treated to tolerance with external-beam radiation therapy without danger of injury to surrounding structures.

Hilaris and associates [3, 6–12] at Memorial Sloan-Kettering Cancer Center have accrued the largest experience with BT in the treatment of lung cancer. In 1979, Hilaris and Martini [8] reported a series of 470 patients treated with BT over a 20-year period. Local early control was achieved in 80% of patients with stages I and II lung cancer and in 60% in stage III. The 5-year survival of this group, however, was only 7%. A review [11] of this experience published in 1987 analyzed more than 1,000 patients treated with BT between 1949 and 1982. Local control in stage III patients with negative mediastinal nodes approximated 71% and in those with involved regional nodes, 63%. The 2-year survival of this group was 51% with a median survival of 24 months. The major cause of failure in the series was the development of metastatic disease.

The 2-year survival rate in the Henry Ford Hospital experience averaged 40% with a median survival of 14 months. In comparison, series of patients with localized inoperable or incompletely resected lung cancer treated by conventional external-beam irradiation had a 2-year survival rate ranging between 8% and 36% with a median survival averaging 6 to 12.5 months [13–19]. It appears that BT can offer improved survival benefits in select patients compared with external-beam radiation therapy in similar patients.

To date, the effectiveness of BT in the treatment of chest malignancies other than primary lung carcinoma has not been documented. Metastatic lesions to the lung and primary unresectable malignant mediastinal masses accounted for 20.4% of this series. The 12-month and 24-month survival figures in both of these categories compare favorably with those seen in primary lung carcinoma (see Table 2). Log-rank analysis showed no significant difference in survival characteristics between these three groups.

Major survival differences occurred when the tumor mass was excised or debulked in conjunction with BT. Kaplan-Meier analysis of survival with log-rank testing showed that reduction in tumor volume versus implantation alone produced a 32.6% and 19.5% improved survival at 12 and 24 months, respectively (see Fig 2). Similar survival benefits were seen when lymph node beds were excised and irradiated with afterloading catheters (see Fig 3). As a consequence, efforts are made at each BT procedure to reduce the tumor burden to the smallest volume by the safest debulking maneuver. No significant improvement in survival was seen according to the cell type of tumor, the stage of primary lung carcinoma, or the type of BT used.

At initial presentation, up to 50% of patients with lung cancer will have far-advanced disease precluding a surgical approach. Only 15% to 20% will have limited disease with potential surgical curability. In the remaining 30% to 35% who have advanced lesions (T3 or T4 tumors or limited malignant mediastinal adenopathy), conventional external-beam radiation therapy is often used. Some of these patients may benefit from an aggressive program of debulking and BT. Current indications for the use of this modality could include implantation of unresectable disease found at the time of thoracotomy performed with curative intent, ^{192}Ir afterload irradiation of resected malignant lymph node beds, implantation of lung malignancies in patients with limited pulmonary function where resection would be hazardous, and good-risk patients with chest malignancies in whom other conventional therapy modalities have failed.

To date, no prospective randomized studies comparing BT with conventional external-beam radiation therapy have been published. Most institutions employing this type of interstitial irradiation generate small numbers of patients, and this precludes randomization. Until these comparisons are made, the true value of BT in the management of chest malignancies will not be known.

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DISCUSSION

DR ROBERT J. GINSBERG (Toronto, Ont, Canada): The report from the Henry Ford Hospital discusses the use of BT, but the presentation we have just heard does not indicate that all these patients also had substantial external-beam irradiation, often postoperatively. Therefore, the report really discusses the use of BT as an adjunct to external-beam irradiation in the management of a variety of thoracic malignancies, the majority of which were lung cancer and the majority of which were debulked.

There appear to be three indications for BT as reported by Lewis and associates. One is to treat residual microscopic or suspected microscopic disease after resection. Another is to treat gross residual disease after a failed attempt at total resection. The third is a planned approach for inoperable disease involving debulking, BT, and external-beam irradiation if it was not given preoperatively.

In their report, Lewis and associates claim improved results compared with those with conventional external-beam therapy as reviewed in the literature. There is no doubt that BT as used by the Memorial Sloan-Kettering and Henry Ford groups can effectively increase the total radiation dose with minimal adverse effects. However, there is still no convincing evidence that this aggressive treatment improves quality or length of life compared with more conventional external-beam treatment.

Before BT becomes an accepted adjuvant modality to improve local disease control, there is a pressing need for a properly designed trial comparing standard treatment, external-beam irradiation, with the more aggressive treatment of debulking, BT, and external-beam irradiation.

I have a question for Dr Lewis. Of the surviving patients who did not have local recurrent disease evident, what percentage had only microscopic or suspected microscopic residual disease at the time of treatment?

DR SHERWYN E. WARREN (Park Ridge, IL): I congratulate Dr Lewis and associates for bringing to our attention a very intriguing and appealing mode of therapy.

One problem that I have had is trying to coordinate treatment with the radiation therapy department. Usually I am unable to have radioactive iodine–tagged seeds available in the operating room when I find an unresectable cancer at thoracotomy.

When the patient is known to have an advanced, unresectable malignancy, our radiotherapists pretreat the patient with irradiation, and this usually precludes further surgical treatment.

Dr Lewis, how do you coordinate intraoperative BT with the radiotherapist, how do you know the quantity of radioactive material you will need for each patient, and how willing are the radiotherapists to deliver radioactive seeds to the operating room?

DR DOUGLAS J. MATHISEN (Boston, MA): I have one question for Dr Lewis. Would you explain how your institution decides which patients are suitable for BT as opposed to conventional radiation therapy?

DR THOMAS M. DANIEL (Charlottesville, VA): I have an answer to the problem of encountering unresectable tumors or lymph nodes at the time of operation and coordinating treatment with the radiation therapy department. In the September 1989 issue of *American Journal of Surgery*, my colleagues and I reported a technique we have developed by which we obtain the afterloading catheters without foreknowledge of the radiotherapists, insert them through the chest tubes in such a way that they fan out and are not obstructed, and then notify the radiotherapists after the fact. They can then take the patient to their facility and use their scanning devices to calculate the dose needed. Within 48 hours, you can place the radioactive substances and accomplish the same goal as if the radiation therapy department had known about the treatment beforehand.

DR HOWARD S. BROWN (Atlanta, GA): I am a little confused about the philosophy of what I have just heard. I have always thought that complete resection of non–small cell carcinoma of the lung is the goal. Are we talking about opening up patients with obviously unresectable disease and administering BT, or are we talking about patients whom we find have inoperable disease and BT is what we have to offer them once they have already been opened?

DR LEWIS: Dr Ginsberg, thank you for your comments. To answer your question about which patients had residual disease, all patients with ^{125}I-labeled implants had gross residual disease. The catheters with ^{192}Ir-tagged sources were left behind for potential microscopic disease in resected lymph node beds or areas of chest wall or mediastinal tumor resection. We do not leave the operating room with uncertainty because we obtain intraoperative pathological confirmation by frozen section.

I agree that many of the issues discussed this afternoon could be answered by prospective randomized series. Certainly Memorial Sloan-Kettering Cancer Center has the largest series. That group has well over 1,000 patients, and Dr Hilaris publishes periodic reviews with data fairly similar to what I have presented today.

Dr Warren, our institution has a large volume of ^{125}I-labeled implants for prostatic carcinoma. Therefore, there seems to be a good stockpile on hand most of the time. I am told by our radiotherapists that the seeds can be ordered and delivered within 24 hours.

How can we predict when we are going to need radioactive seeds? In about half of our patients, BT is performed on a planned basis. Many of the remaining patients will have some physical or radiographic abnormality that raises the possibility of implantation. Even though the seeds may not be placed, use in other areas such as prostatic cancer would ensure they are not wasted.

To determine the number of seeds required for implantation, we use the nomogram published by the Memorial Sloan-Kettering Cancer Center. Through preoperative staging, we have a good idea of how many seeds will be required. In the operating room, we measure the tumor carefully and base our implant volume on these values.

How do we select patients for BT? In the initial part of the series, this modality was used when an unexpected unresectable situation was encountered during operation. Because our initial survival figures were good, we offered this modality to patients who had preoperative chemotherapy or radiation therapy or both and literally had nothing else open to them. About half of our patients fall into that category.

Concerning long-term survival, we have 10 of 103 patients surviving more than 36 months and 1, past 60 months. However, the final value of this modality of therapy will not be known until there is a good randomized series. I certainly would welcome that.

Notice From the Southern Thoracic Surgical Association

The Thirty-seventh Annual Meeting of the Southern Thoracic Surgical Association will be held at the Hyatt Regency Cerromar Beach, Puerto Rico, November 8–10, 1990. There will be a $155 registration fee for nonmember physicians except for guest speakers, authors and coauthors on the program, and residents. There will be a $50 registration fee for attendees of the Postgraduate Course, which will be held the morning of Thursday, November 8, 1990. The Postgraduate Course will provide in-depth coverage of thoracic surgical topics selected primarily as a means to enhance and broaden the knowledge of practicing thoracic and cardiac surgeons.

Members wishing to participate in the Scientific Program should submit an original abstract and one copy by May 15, 1990, to Donald C. Watson, Jr, MD, Program Chairman, Southern Thoracic Surgical Association, 111 East Wacker Drive, Chicago, IL 60601. Abstracts must be submitted on the Southern Thoracic Surgical Association abstract submission form. These forms may be obtained from the Association's office. All slides used during the presentation must be 35 mm. Manuscripts of accepted papers must be submitted to *The Annals of Thoracic Surgery* prior to the 1990 meeting or to the Secretary-Treasurer at the opening of the Scientific Session.

Applications for membership should be completed by July 1, 1990, and forwarded to Lenox Baker, Jr, MD, Membership Committee Chairman, Southern Thoracic Surgical Association, 111 East Wacker Drive, Chicago, IL 60601.

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Correction of Total Anomalous Pulmonary Venous Connection Below the Diaphragm

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Six infants with total anomalous pulmonary venous connection below the diaphragm had correction by modification of conventional surgical technique. Catheterization revealed the confluence of the pulmonary veins draining into a descending vein below the diaphragm. Symptoms of pulmonary venous hypertension and low cardiac output were typical. All had repair with circulatory arrest (average time, 32 minutes). Mobilization of the pulmonary veins and the descending vein is important. The descending vein was transected at the diaphragm. Its anterior surface was incised through the confluence of the pulmonary veins, thus creating an open Y incision. This large Y-shaped vein was anastomosed to the left atrium and carried obliquely to the tip of the left atrial appendage. The anastomosis was fashioned so that the long limb of the Y rotated anteriorly and superiorly to substantially enlarge the left atrium, making the total diameter of the anastomosis larger than the mitral valve orifice. This simplified the surgical repair and allowed direct suture closure of the atrial septal defect in all patients, as the left atrial size was at least doubled. No postoperative complications occurred, and the patients were discharged an average of 4.2 days postoperatively. Restudy at an average of 3.5 years revealed normal pressures and normal architecture by angiography. Use of the descending vein as an integral part of the reconstruction and enlargement of the left atrium was the major technical factor leading to a successful outcome in these patients and eliminating a patch or transposition of the atrial septum.

(Ann Thorac Surg 1990;49:734–9)

Major improvement in the morbidity and mortality of patients with total anomalous pulmonary venous connection below the diaphragm has been achieved during the past three decades [1–5]. Most of these advances have been attributed to earlier diagnosis and operative intervention, improved techniques of circulatory arrest, and better perioperative management [6–14]. Operative techniques for repair of total anomalous pulmonary venous connection below the diaphragm have changed minimally, even though different surgical methods have been employed [14–21]. A variety of techniques have been recommended for anastomosis of the common pulmonary vein to the left atrium [1, 8, 16, 21, 22]. The goal of surgical intervention should be the creation of a large left atrium with a widely patent pulmonary venous to atrial anastomosis that is equal to or greater than the orifice of the mitral valve. This report describes a technical modification used in 6 consecutive infants who had correction of total anomalous pulmonary venous connection below the diaphragm.

Material and Methods

Study Population

From 1979 to 1987, 23 infants with all varieties of anomalous pulmonary venous drainage were seen. The cases of 6 neonates having surgical correction of total anomalous pulmonary venous connection below the diaphragm were analyzed. The diagnosis was made on clinical grounds and confirmed by echocardiography and cardiac catheterization. These studies typically demonstrated the confluence of the pulmonary veins draining into a descending vein below the diaphragm. Dynamic angiograms demonstrated the very slow flow of contrast medium through the lungs (Fig 1). The clinical course and hemodynamics

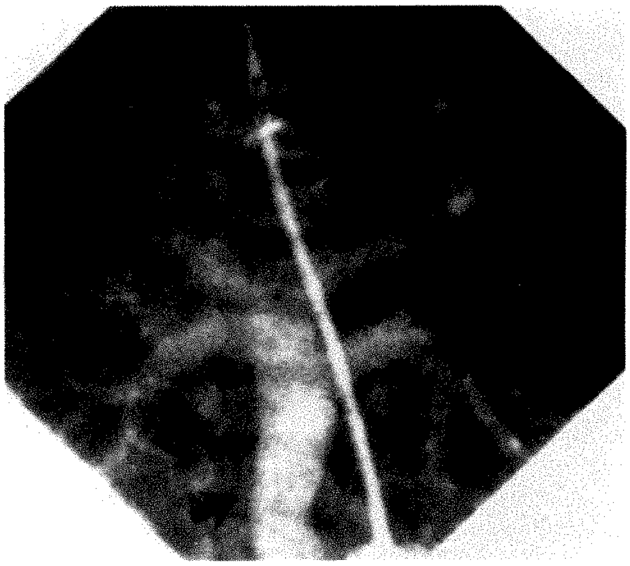

Fig 1. Preoperative angiogram demonstrating the confluence of the pulmonary veins into a descending vein (arrow).

Presented at the Twenty-fifth Anniversary Meeting of The Society of Thoracic Surgeons, Baltimore, MD, Sep 11–13, 1989.

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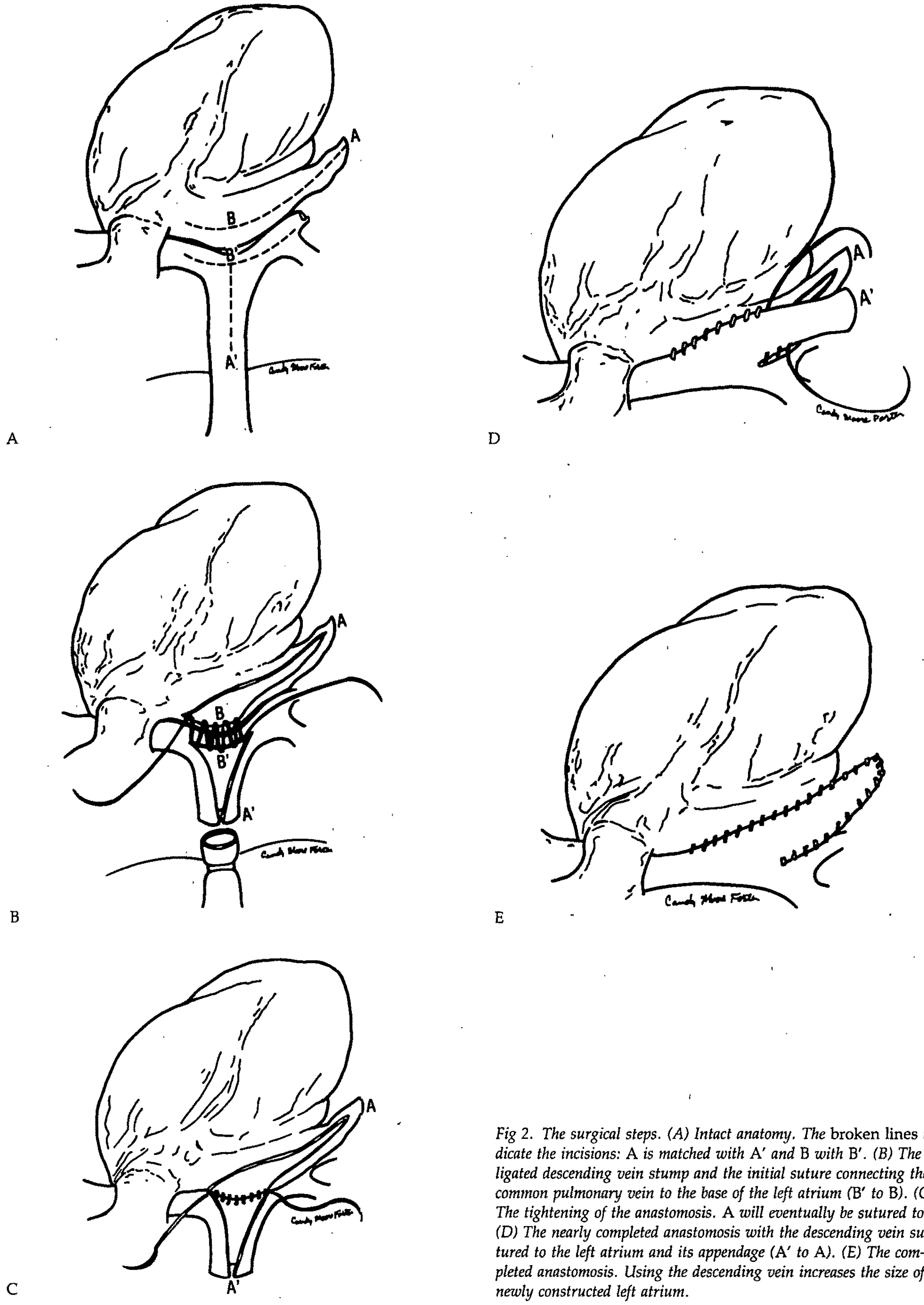

Fig 2. The surgical steps. (A) Intact anatomy. The broken lines *indicate the incisions:* A *is matched with* A′ *and* B *with* B′*. (B) The ligated descending vein stump and the initial suture connecting the common pulmonary vein to the base of the left atrium* (B′ *to* B)*. (C) The tightening of the anastomosis.* A *will eventually be sutured to* A′*. (D) The nearly completed anastomosis with the descending vein sutured to the left atrium and its appendage* (A′ *to* A)*. (E) The completed anastomosis. Using the descending vein increases the size of the newly constructed left atrium.*

were typical with signs and symptoms of pulmonary venous hypertension and low cardiac output.

Age at operation ranged from 24 to 37 hours (mean age, 30 hours). Weight ranged from 2.1 to 3.7 kg (mean weight, 3.2 kg). All patients had situs solitus. They underwent operative repair an average of six hours from the time of diagnosis. Late follow-up included echocardiography and cardiac catheterization.

Operative Technique

The patients underwent surgical repair with topical hypothermia, core cooling, and circulatory arrest. All repairs were performed through a mediastinotomy when the rectal temperature reached 32°C with topical cooling.

The ductus arteriosus is routinely ligated. Cardiopulmonary bypass is established using a single venous drainage cannula in the right atrium with arterial return through the ascending aorta. Body temperature is further reduced to 18°C with cardiopulmonary bypass. A mixture of nitroglycerin and sodium nitroprusside is infused during the cooling period with the aim of achieving maximal vasodilatation and reducing temperature gradients. Total circulatory arrest allows the venous cannula to be removed to obtain optimal exposure.

During the cooling phase, the apex of the heart is lifted toward the patient's chin, and the anomalous venous connection is identified. Extensive circumferential mobilization of both right and left pulmonary veins, along with their confluence into the descending vein, is carried out. The descending vein is mobilized down into its diaphragmatic hiatus to obtain as much length as possible.

After mobilization of the intrapericardial pulmonary venous system has been completed, the descending vein is gently "tented" up through its diaphragmatic hiatus and suture ligated. The vein is transected and the distal stump allowed to retract below the diaphragm. The other end of the descending vein is left open during the remaining cooling period to allow the pulmonary venous system to decompress into the pericardial well. It took approximately six to eight minutes to cool the neonates from 32° to 18°C. During this time, the surgical maneuvers just described were performed.

At 18°C, total circulatory arrest is employed to achieve a dry and quiet operative field. The venous cannula is removed, and a small right atriotomy is performed to expose the atrial septal defect. The descending vein is incised longitudinally along its anterior wall into the confluence of the right and left pulmonary veins.

After the apex of the heart has been gently elevated, the now mobile pulmonary venous structures can be lifted toward the elevated heart. The heart is slowly allowed to relax back into the pericardium so that the left atrial incision can be matched to the incision in the pulmonary veins. This allows the anastomosis to be performed with minimal kinking or twisting. The initial incision in the left atrium is near its base adjacent to the atrial septum. The incision is eventually carried obliquely toward and into the left atrial appendage. The entire atrial incision is not completed initially, but extended periodically with the anastomosis in progress. This allows optimal matching of the vein opening to the atriotomy.

Table 1. Preoperative and Postoperative Catheterization Data[a]

Pressure	Preoperative	Postoperative[b]
Right atrium	8.5 ± 2.5 (6.0–11)	7 ± 3 (4–10)
Right ventricle	83/3 ± 7.0 (75/1–90/6)	30/6 ± 4 (25/2–35/5)
Pulmonary artery	88/46 ± 11 (77/40–99/50)	27/15 ± 4 (22/10–33/18)
Aorta	54/44 ± 7 (46/35–60/49)	85/54 ± 8 (78/50–92/56)
Left ventricle	72/6 ± 5 (68/4–77/8)	85/5 ± 5 (79/0–90/4)

[a] Data are for 5 patients and are shown as the mean ± the standard error of the mean in millimeters of mercury with the range in parentheses.
[b] The average follow-up is 3.5 years (range, 1 to 7 years).

The anastomosis is performed with 7-0 Prolene sutures (Ethicon, Somerville, NJ) and optical magnification. The first group of sutures is placed parachute fashion, as for a coronary anastomosis. The base of the atrial incision is sewn to the pulmonary venous confluence at the point where they match. The suture line is completed around both borders of the pulmonary venous confluence and then is continued into what had been the descending vein. The descending vein is rotated superiorly as it is matched and sutured to the continued incision in the left atrium. The suture line is completed in the atrial appendage. Figure 2 summarizes the surgical steps. Total circulatory arrest time averaged 32 minutes (range, 28 to 37 minutes).

Cardiopulmonary bypass is then reestablished for rewarming. With rewarming, the ice packs, which had remained on the infant's head during the circulatory arrest phase, are removed, and the vasodilator drip is restarted. Also during rewarming, the suture line can be examined for anastomotic leaks. Temporary pacing wires are placed and standard operative maneuvers performed. At 36°C, the patient is removed from cardiopulmonary bypass, and the operation is completed in routine fashion.

Results

All patients survived and were discharged an average of 4.2 days postoperatively. Postoperative follow-up to 7 years demonstrated regular sinus rhythm, normal pulmonary artery pressure, and good cardiac growth and function. Table 1 summarizes the pertinent preoperative and postoperative catheterization data. Though Prolene was used as the continuous suture for the anastomosis, no late venous obstruction occurred (Fig 3). One patient had early reoperation (2 months) for pulmonary venous obstruction of the right pulmonary veins. This technical error was repaired, and the patient recovered uneventfully.

Comment

Anomalous pulmonary venous drainage is a malformation in which the communications between the pulmo-

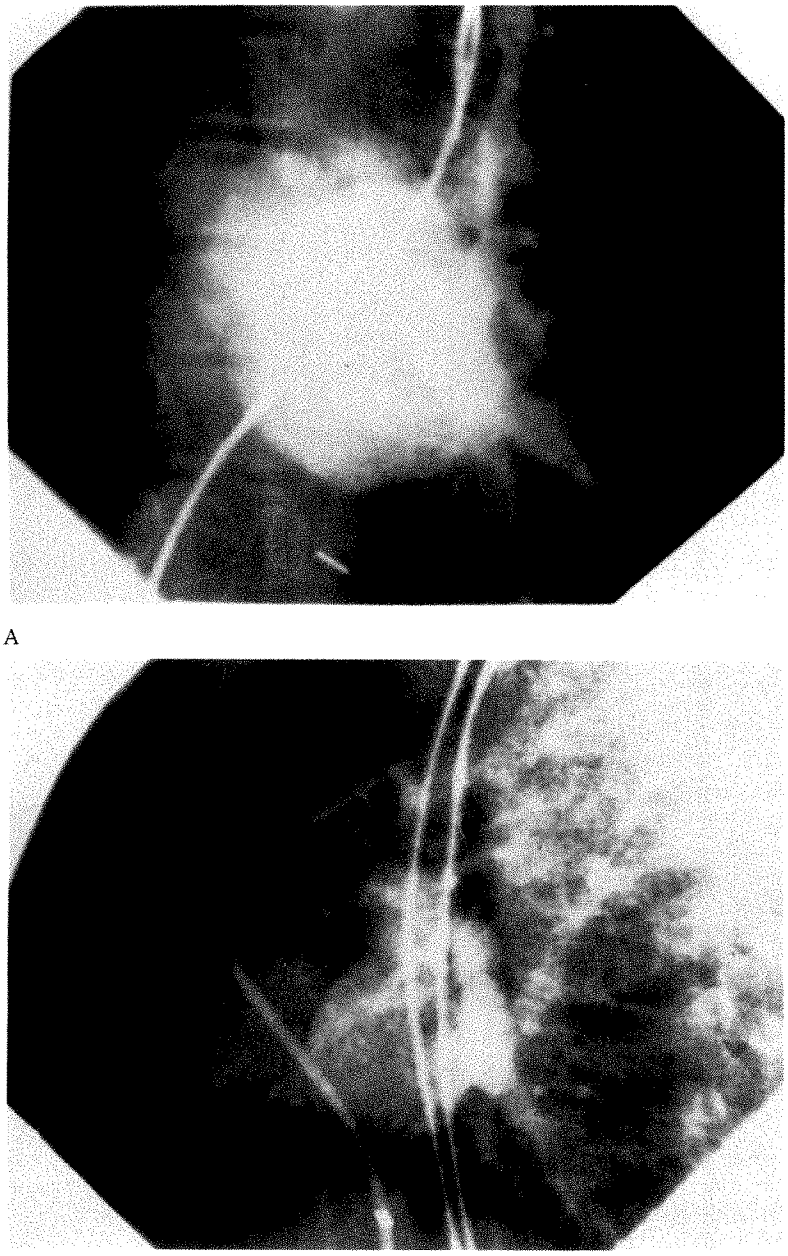

Fig 3. Angiograms made 7 years after correction of total anomalous pulmonary venous connection below the diaphragm with continuous 7-0 Prolene suture. (A) Large left atrium and (B) left atrium and ventricle (arrow).

nary venous plexus and the left atrium fail to develop [14]. This defect results from the failure of incorporation of the common pulmonary vein into the left atrium, and causes the venous drainage of the lung to be dependent on the persistence of other anatomical components of the fetal venous system [2, 6, 10, 11, 13, 14]. The loss of this tissue, which remains in the anomalous pulmonary venous system, results in a left atrium that is smaller than normal. One aspect of surgical correction should be aimed at replacing as much of the deficient tissue as possible. Freeing up the descending vein and incorporating it into the new left atrium achieves the goal of constructing a normal-sized left atrium. The added tissue provided by the descending vein permits the creation of an anastomosis as large as or larger than the mitral orifice. In addition, using the "freed" descending vein allows the creation of a "mobile," kink-free, patent anastomosis. We recommend that other surgeons consider using the technique described.

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DISCUSSION

DR EDWARD J. BOVE (Ann Arbor, MI): Total anomalous pulmonary venous connection below the diaphragm represents approximately one third of all cases of total anomalous connections. These infants generally are seen within the first few days of life and are in poor condition with respiratory distress, hypoxemia, and acidosis. Dr Phillips and his colleagues describe 6 consecutive neonates with infracardiac total anomalous drainage that was repaired between 1979 and 1987. The neonates underwent operation at an average age of 30 hours using standard methods of deep hypothermia and circulatory arrest. In each case, the anastomosis of the pulmonary venous confluence to the left atrium was augmented with the opened descending vertical vein, which was tailored up to the left atrial appendage.

Phillips and colleagues have achieved the enviable result of no early or late deaths. Even more remarkable, hospital discharge was possible at an average of only 4.2 days after operation. One patient, however, required reoperation 2 months later to revise a technical error causing obstruction to the right pulmonary veins.

Although early reports suggested that left atrial size in patients with total anomalous connections may be smaller than normal, this is not generally considered true. The apparent smallness is, in fact, due to the lack of incorporation of the pulmonary venous confluence. Therefore, repair establishes a normal-sized left atrium. Further, late studies have documented satisfactory anastomotic growth and excellent late results with the exception of those 5% to 10% of patients in whom pulmonary venous stenosis develops apart from the actual suture line.

The orientation of the pulmonary vein confluence in infracardiac total anomalous pulmonary venous connection is vertical or tree shaped. Often the pulmonary veins themselves are situated quite inferiorly below the level of the left atrium. Mobilization of the veins, particularly the descending vertical vein, well below the level of the diaphragm is a most useful maneuver. It allows the confluence to reach the left atrium and augments the total circumference of the anastomosis, thus reducing the incidence of late obstruction.

One of our patients had the typical tree-shaped formation of total anomalous pulmonary venous drainage below the diaphragm. The left atrium in this patient was considerably above the confluence. A technique in which the descending vertical vein was turned up as a flap, similar to what Dr Phillips described, led to excellent late results.

I have three questions for Dr Phillips. First, what was the mechanism of obstruction in the patient who required reoperation? Was it secondary to kinking of the anastomosis?

Second, you presented a rather horizontal pulmonary venous confluence, not the more typical vertical one described in most reports. Does this alter your technique, and do you incise the pulmonary veins directly into their branches to enlarge the anastomosis?

Finally, the early discharge from the hospital is remarkable. In our series, many of these patients are seen with severe pulmonary edema and have required prolonged ventilatory support. For these patients, would you recommend extracorporeal membrane oxygenation either preoperatively or postoperatively?

DR GERHARD ZIEMER (Hannover, West Germany): I congratulate Dr Phillips for using this technique primarily. Although my colleagues and I occasionally incised the left atrial appendage in patients with a diminutive left atrial back wall, we did not do so routinely. In one redo procedure, we had to do it for late anastomotic stenosis.

We absolutely agree that efforts should be made to avoid foreign material. However, in patients with total anomalous pulmonary venous drainage with what I would call an "interpulmonary venous long-distance confluence," extensive mobilization is not advisable. In such patients, the lower right and left pulmonary veins merge to the confluence at the diaphragmatic level, leaving almost no supradiaphragmatic length to the confluence. Any extensive dissection of the lower pulmonary veins and inferior confluence carries a high risk of obstructing these structures if rotated. We simply clip the confluence in situ at the level of the diaphragm and then incise it, usually into the right upper vein and, if necessary, into the left side. We often place an intraatrial Gore-Tex patch. It not only enlarges but also stents the anastomosis. What do you do in such patients?

DR RONALD C. ELKINS (Oklahoma City, OK): I have one note of caution. I have been following children with this entity for about 18 years and have operated on some 70 of them. Of those 70, 3 children, 1 at 7, 1 at 10, and 1 at 11 years of age, required reoperation because of scarring related to the circumferential Prolene anastomosis, which produced pulmonary venous hypertension. I think that with the suture material available, there are better choices than Prolene for this anastomosis.

DR PHILLIPS: Dr Bove, the obstruction of the right pulmonary veins in 1 patient was the result of a technical error. That was the only patient in whom we did extend the incision into the vein directly, and the suture caught the posterior wall.

As for your second question, these are small secondary veins, and making the anastomosis, even with optical magnification, is somewhat tedious. Therefore, we try to avoid extending any incisions into the secondary veins.

I really have no good explanation for the early discharge. There is no question that these patients were very sick. They had pulmonary venous hypertension. Possibly we are very lucky; possibly the early diagnosis and the very early surgical intervention helped them recover quickly. I do believe that at least in the 6 patients described, the use of this technique permitted a very, very wide anastomosis and relieved any residual pulmonary venous obstruction.

I think Dr Ziemer's comments are very appropriate. The confluence is quite low, though if you are careful, you can rotate up the entire pulmonary venous confluence as well as the descending vein and appropriately position it into the left atrium. We do not necessarily use the entire left atrial appendage, but we increase the incision for a good match as we are doing the anastomosis. In past years I used different techniques and had to patch the atrial septal defects to increase the size of the left atrium, but with this technique we have not had to do that.

I appreciate the comments of Dr Elkins. Seventy patients are quite a few more than 6. We have talked about using absorbable sutures, but the results, at least up to 7 years, have not shown any problem with Prolene.

Aortic Valve Operation After Percutaneous Aortic Balloon Valvuloplasty

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From January 1986 to May 1988, 179 patients underwent percutaneous aortic balloon valvuloplasty at our institution. Forty-five (25%) patients subsequently required operation at this institution or other institutions. The indication for percutaneous aortic balloon valvuloplasty was patient preference in 15 and an estimation of excessive operative mortality in 30. Three patients required emergency operation immediately after valvuloplasty, and the other 42 had an elective operation a mean of 7.8 months afterward. All but 3 of these patients had major progression of symptoms, and all had valvular restenosis. In spite of the high-risk status of these patients, there were only four hospital deaths among the 45 patients. Three additional patients have died a mean of 11.4 months postoperatively. The condition of all survivors remains clinically improved after the valve operations. Percutaneous aortic balloon valvuloplasty must be recognized as a palliative procedure, and should be reserved for patients who are truly not candidates for operation or situations in which a brief period of reduction in gradient might improve a patient's condition before operation.

(Ann Thorac Surg 1990;49:740–5)

With the advent of percutaneous aortic balloon valvuloplasty (PABV) in 1986, an alternative to operative valve replacement became available for the treatment of adult calcific aortic stenosis. It was hoped that PABV would offer a less invasive, and therefore less acutely hazardous, means of treating these patients. As the clinical role for PABV was being defined, the procedure was recommended for elderly patients who were considered to be poor surgical candidates [1–3]. Once the procedure became available, however, some patients and their physicians elected the less invasive option of PABV over aortic valve replacement (AVR).

The clinical use of PABV has evolved despite limited knowledge of the short-term risks and the absence of data regarding the late results. Recent reports [4–6] have indicated that although the early mortality rate of PABV is acceptably low, there is a substantial recurrence of clinically and hemodynamically significant aortic stenosis within 6 to 9 months.

Because our institution has a large experience with PABV, we thought it would be useful to review the results of balloon valvuloplasty from the perspective of the cardiac surgeon. The early experience with many of these patients is detailed in a recent report from our institution [4]. In this report, we examine the patients who underwent PABV at this institution and subsequently required a cardiac operation, either emergently after PABV or electively because of the development of aortic restenosis.

Material and Methods

From January 1986 to May 1988, 179 patients with aortic valve stenosis underwent PABV at Boston's Beth Israel Hospital. Forty-five (25%) of these patients subsequently required a cardiac operation and are the subject of this review. Twenty-one patients underwent cardiac operation at our institution, and 24 were operated on at other hospitals. (During this same 28-month period, we performed 112 AVRs in patients who did not have prior PABV; 41 had AVR only and 71, AVR plus coronary artery bypass grafting [CABG] or mitral valve replacement.) All the patients were followed up for a minimum of 7 months. Table 1 lists the baseline clinical and hemodynamic characteristics of this group of 45 patients at the time of initial PABV.

Each patient's chart (from initial PABV and subsequent operation if performed at our institution) was reviewed, and all surviving patients were interviewed. The indication for PABV was determined from these sources. A single primary reason for the use of PABV was ascertained in each instance. Thirty patients (67%) were considered poor candidates for cardiac operation with a high estimated perioperative mortality. Fifteen patients (33%) elected PABV. The assessment of increased operative risk was made by the referring physician, the cardiologist, or a cardiac surgeon at another institution for 43 of the 45 patients.

The hemodynamic data were determined from cardiac catheterization. Data before and immediately after PABV were available for all patients. Twenty-seven patients were restudied at this institution before cardiac operation, and the cardiac catheterization data were collected and analyzed.

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Table 1. Baseline Characteristics of the 45 Patients

Variable	No. of Patients[a]
Age (yr) (mean ± standard deviation)	71 ± 12
Men:women	24:21
Symptoms	
Angina/CHF	39
NYHA class II	1
NYHA class III/IV	38
Syncope	6
Coronary artery disease	
Prior MI	4
1 Vessel	13
2 Vessels	3
3 Vessels	3
Prior CABG	3
Mitral valve disease	
Mitral stenosis	4
Mitral regurgitation	2

[a] All data are shown as number of patients except age.

CABG = coronary artery bypass grafting; CHF = congestive heart failure; MI = myocardial infarction; NYHA = New York Heart Association.

Table 2. Indications for Percutaneous Aortic Balloon Valvuloplasty

Indication	No. of Patients
Poor surgical candidate	30/45
Age ≥ 80 yr ± CAD	14
LVEF ≤ 0.35	6
Prior CABG	3
Prior mediastinal irradiation	1
Severe pulmonary disease	1
Prior attempt at operation	2
Anticoagulation contraindicated	1
Salvage valvuloplasty[a]	1
Planned urgent, extensive noncardiac-related procedure	1
Patient preference	15/45

[a] This means that a patient with mild to moderate aortic stenosis and severe coronary disease experienced extreme hemodynamic instability with percutaneous transluminal coronary angioplasty and had attempted percutaneous aortic balloon valvuloplasty while awaiting transfer to the operating room.

CABG = coronary artery bypass grafting; CAD = coronary artery disease; LVEF = left ventricular ejection fraction.

The postoperative clinical status of all patients was ascertained by patient interviews. All cardiac symptoms were recorded. The clinical status of the patients before and after PABV and before and after cardiac operation was determined from these interviews and from comparison with preexisting data.

In the interest of accuracy, the data relating to perioperative morbidity are reported only for the patients who underwent cardiac operation at this institution. Follow-up clinical status was obtained for all operative survivors without regard to the institution in which the operation was performed.

Results

The characteristics of our patient population are shown in Table 1. The mean age of the 45 patients was 71 ± 12 years with a range of 45 to 88 years. Sixteen (36%) of the patients were more than 80 years old at the time of initial PABV. Coronary artery disease was present in 19. Four patients had a history of previous myocardial infarction, and 3 patients had a history of previous CABG. Mitral stenosis was documented in 4 patients, 2 of whom underwent successful percutaneous mitral balloon valvuloplasty in addition to PABV. Two patients exhibited evidence of moderately severe mitral regurgitation by left ventriculography. Six patients had ejection fractions of less than 0.35. The mean aortic valve area for the 45 patients was 0.60 ± 0.20 cm^2.

One third of the patients preferred PABV to cardiac surgical intervention. Two thirds of the patients undergoing initial PABV were considered poor surgical candidates. The majority of them were considered high risks because they were elderly with associated medical illnesses or they had poor left ventricular function (ejection fraction ≤ 0.35) or both. Technical considerations were important in 6 patients. Three patients had prior CABG; 1 patient had a history of exposure to high-dose mediastinal irradiation for treatment of lymphoma; and 2 patients (previously operated on at other institutions) had had attempts at AVR aborted because of extensive calcification of the aorta. The indications for PABV are listed in Table 2.

Percutaneous aortic balloon valvuloplasty was successfully completed in 44 of the 45 patients and resulted in an increase in the calculated aortic valve area (Table 3). On restudying 27 patients at a mean of 11 months, the mean aortic valve area had decreased to a value similar to that before the procedure. The mean transaortic gradient increased, and cardiac index decreased (see Table 3).

Table 3. Hemodynamic Changes Before and After Valvuloplasty but Before Operation

Variable	Before PABV[a] (n = 45)	After PABV[a] (n = 44)	Follow-up (n = 27)
Aortic valve area (cm^2)	0.60 ± 0.20	0.95 ± 0.40	0.65 ± 0.25
Mean transaortic gradient (mm Hg)	57 ± 19.0	34 ± 13.0	50.0 ± 15.0
Cardiac index ($L/min/m^2$)	2.7 ± 0.68	2.95 ± 0.72	2.6 ± 0.72

[a] Significance: $p < 0.001$ for comparisons of each variable between Before PABV and After PABV and between After PABV and Follow-up.

PABV = percutaneous aortic balloon valvuloplasty.

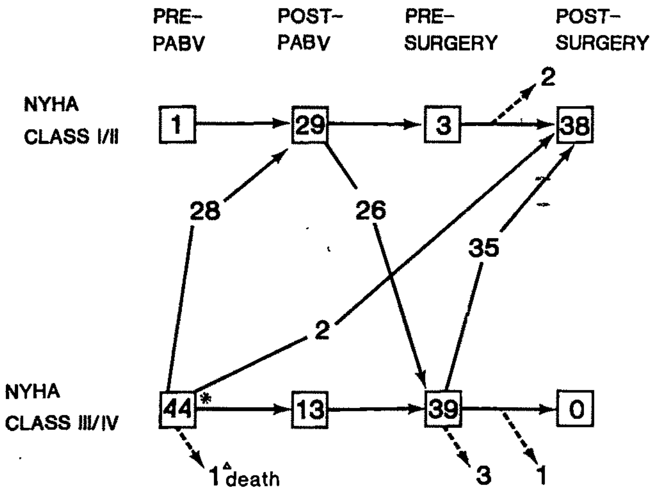

Fig 1. Changes in the clinical status of the patient population. The clinical condition is noted before percutaneous aortic balloon valvuloplasty (PRE-PABV), *immediately after the procedure* (POST-PABV), *immediately before operation* (PRE-SURGERY), *and at the time of follow-up for this review* (POST-SURGERY). *Deaths are indicated by the* broken lines *and are detailed in the text. The mean interval between POST-PABV and PRE-SURGERY was 7.8 months and between PRE-SURGERY and POST-SURGERY, 11 months. One patient could not have a clinical status evaluation after valvuloplasty because he sustained a left ventricular perforation and pericardiocentesis and was taken to the operating room. He died shortly after operation (Δ). Two patients had valvuloplasty and survived emergency aortic valve replacements (*). They had their next assessment in the POST-SURGERY category.* (NYHA = *New York Heart Association.)*

Table 4. Cardiac Surgical Procedures Performed

Procedure	No. of Patients
AVR	24
AVR + CABG	14
AVR + CABG + MVR	1
Open aortic valvuloplasty	1
Redo CABG + AVR	3
Redo AVR (prior sternotomy only)	2[a]

[a] These 2 patients had previous attempts at AVR at other institutions aborted when extensive aortic calcification was found at operation.

AVR = aortic valve replacement; CABG = coronary artery bypass grafting; MVR = mitral valve replacement.

After PABV, 28 patients (67%) experienced improvement in symptoms from New York Heart Association class III/IV to class I/II. Nearly one third (13 patients), however, had persistent symptoms of moderate severity. In this small cohort of 45 patients who ultimately required valve operations, we could not identify any correlation between an achieved valve area greater than 1.0 cm^2 and subsequent clinical improvement. However, patients in this study often had concomitant cardiac problems (coronary artery disease or mitral valve disease), which would make even a satisfactory reduction in the transvalvular aortic gradient less likely to achieve elimination of all symptoms.

Figure 1 illustrates the functional classification before PABV, after PABV, immediately before operation, and at late follow-up. No interview after PABV was possible with 1 patient who died within hours of an emergency operation performed immediately after attempted valvuloplasty. Two other patients underwent emergency operations and hence had no assessment after PABV; they survived and did have late postoperative follow-up. Twenty-six of the 28 patients whose symptoms improved after PABV showed subsequent deterioration in clinical status. After a mean follow-up of 7.8 ± 1.0 months, 39 of the original 44 patients in New York Heart Association class III/IV were again in that category. The 3 patients who showed no clinical deterioration had evidence of hemodynamically significant restenosis on follow-up catheterization.

There were 41 hospital survivors after operation, an operative mortality of 8.9% (4/45). Table 4 lists the types of procedures performed. Three patients underwent emergency cardiac operation resulting in one hospital death, and 42 patients underwent elective cardiac operation with three hospital deaths. Both patients who had had prior aborted aortic valve operations survived. The hospital deaths are detailed in Table 5. The patient who died of a consumption coagulopathy had reinfusion of his pericardial blood for two hours. This unusual measure was undertaken in an effort to stabilize his condition until an operating room became available.

Only the 18 patients who survived cardiac operations at this institution were studied for perioperative morbidity. The complications occurring in the patients who died in the postoperative period are noted in Table 5. Exclusive of those patients, 2 patients sustained a perioperative myo-

Table 5. Hospital Deaths After Cardiac Operation

Patient No.	Age (yr)	Sex	Indication for initial PABV	Indication for Operation	Procedure Performed	Cause of Death
1	79	M	Poor surgical candidate	Acute cardiac tamponade	AVR + CABG, repair of LV perforation	Postoperative coagulopathy
2	70	F	Poor surgical candidate	Restenosis	AVR	Stroke, respiratory failure
3	66	F	Poor surgical candidate	Restenosis	AVR	Stroke
4	37	M	Patient preference	Restenosis	AVR	Postoperative cardiac arrest

AVR = aortic valve replacement; CABG = coronary artery bypass grafting; LV = left ventricular; PABV = percutaneous aortic balloon valvuloplasty.

cardial infarction as defined by cardiac enzyme elevations, and 1 patient had limb ischemia due to femoral artery injury after femoral artery cannulation. This was successfully treated with open vascular repair. The mean postoperative length of stay was 15.2 days (range, seven to 54 days).

Late follow-up revealed that 38 patients had persistent improvement in clinical status after valve replacement at a mean of 11 ± 7.5 months (see Fig 1). There were three late deaths among the 41 hospital survivors. The condition of 1 patient with associated triple-vessel coronary artery disease and mitral regurgitation did not improve clinically after AVR and CABG. This patient died in the eighth postoperative month of congestive heart failure. Two patients experienced substantial clinical improvement before their late deaths. One died 9 months after operation of complications of chronic mediastinal infection, and the other died 21 months after operation of a urinary tract malignancy.

Comment

An aging population has resulted in a growing number of elderly patients with severe symptomatic calcific aortic stenosis [7]. The prognosis with conservative medical therapy for elderly patients with severe, symptomatic aortic stenosis is poor, with 1-year and 2-year survival rates of 57% and 37%, respectively [8]. Yet these patients, who often have multiple medical problems, can face a high risk with cardiac surgical intervention. Percutaneous aortic balloon valvuloplasty has been proposed as an alternative for the treatment of aortic stenosis.

Percutaneous aortic balloon valvuloplasty can be performed with low early mortality rates (3.5% to 9%), even in the elderly. Short-term follow-up of between 5.5 and 9.0 months has been reported for patients surviving successful PABV. The mortality rate during this period of follow-up is 16% to 28%, and 28% to 36% have evidence of clinical recurrence of symptoms [4–6]. Assuming that the late deaths are related to the valvular disease, the restenosis rate after successful PABV at 6 to 9 months is probably as high as 50%. Furthermore, hemodynamically demonstrable restenosis can develop without recurrence or worsening of symptoms [6, 9, 10]. Three of 45 patients in this study showed no clinical deterioration despite hemodynamic evidence of aortic valve restenosis, a finding underscoring the necessity for continued echocardiographic follow-up.

Assessment of operative risk in this subset of patients is difficult. Published operative mortality rates for myocardial revascularization, valve replacement, and combined procedures in patients older than 70 years range from 3.1% to 18% [11–13]. Edmunds and associates [14] reported a cardiac surgical mortality rate in octogenarians of 29%. The number of deaths was highest in octogenarians with class IV symptoms, previous myocardial infarction, the need for emergency operation, or cachexia. When these risk factors were excluded, the early operative mortality rate was still 15%. In another series, Tsai and co-workers [12] reported a 13% early surgical mortality rate for cardiac operations in octogenarians. The highest mortality was among patients with class IV symptoms and among those requiring emergency surgical intervention.

In a recent study by Bessone and colleagues [15], 219 patients with an average age of 75 years had a hospital mortality rate of 12% for AVR alone and 8.9% for combined AVR and CABG. The long-term survival at 7 years was 77% for patients who had AVR and 57% for patients who had AVR and CABG. Di Lello and co-workers [16] reviewed 430 aortic valve operations and found that death was significantly related to age (greater than 55 years), New York Heart Association class III/IV, and the presence of coronary artery disease.

Our patients were certainly at increased risk for operative death. Forty-four of the 45 patients were in New York Heart Association functional class III or IV, 16 were octogenarians, 5 had previous cardiac operations, 18 required combined procedures, and 3 required emergency operations. Yet the hospital mortality rate in this "high-risk" group was 8.9% (4/45). All 16 octogenarians survived the procedure. Certainly the determination of high risk based primarily on age greater than 80 years is unfounded in our experience. We do recognize, however, that the PABV may well have served to select out a more hardy "high-risk" population. The mean age of our patients having operation was 71 years, whereas the mean age of the entire group of PABV patients was 77 years. The early death rate after PABV undoubtedly eliminated patients who might have been less likely to survive operation. There have been only three late deaths among the 41 hospital survivors. The survivors have continued to show clinical improvement at a mean follow-up of 11 months.

It is unrealistic to directly compare the results of PABV with operative valve replacement. The PABV group includes some patients who would not be considered operative candidates under any circumstances. The 3.5% early mortality rate of PABV [4] offers an attractive, and occasionally unique, opportunity to such patients. The late results (PABV late mortality rate of 16% to 28% at a few months compared with our late operative mortality rate of 7.3% at a mean of 11 months), however, make it important to reassess patients after PABV. Importantly, the magnitude of hemodynamic changes associated with PABV did not correlate with the time to operation. For this reason, it seems particularly difficult to predict, immediately after PABV, who will require AVR and when. If a patient becomes an acceptable candidate for operation, he or she should be operated on during the interval of palliation, rather than wait until the likely restenosis. Contrarily, some patients will remain unacceptable candidates for cardiac operations and may be relegated to repeat PABV if possible. The reasons for failure of the PABV procedure have been well delineated [17], and all patients require careful longitudinal follow-up afterward.

Despite the restenosis rate, PABV should not be completely discarded, for it is clearly useful in managing certain patients. We and others [18] have used PABV in patients requiring urgent noncardiac-related operation. Patients who present insurmountable technical problems for open operation should be offered PABV with no option of operative intervention. We are currently investigating the use of PABV as a means of improving the clinical condition for subsequent interval AVR of patients with advanced heart failure, severe pulmonary disease, and low ejection fractions. A hemodynamically successful PABV resulting in clinical improvement readily predicts a successful open operation, perhaps at lesser risk.

Currently, we view PABV as a short-term palliative procedure that should be reserved for the truly high-risk patient or for patients who must show improvement before operation. Risk assessment requires an interdisciplinary evaluation, and should include both cardiology and cardiac surgical input. It is important that patients who "elect" PABV over operation be counseled as to the palliative nature of the valvuloplasty procedure. Patients who are not candidates for operation and who have successful PABV should be reassessed during their interval of clinical improvement. If they are then deemed to have an appropriate operative risk, they should have early AVR.

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DISCUSSION

DR FRANCIS ROBICSEK (Charlotte, NC): This excellent paper shows that the controversy surrounding balloon dilation of the aortic valve is still very much alive. I used the term *dilation* instead of the term *valvuloplasty* because calling the breaking of the aortic valve "plasty" is as appropriate as telling somebody who just had his nose bashed in that he had a "nasoplasty."

The cardiologists document major drops in the aortic gradient during the course of balloon dilation. The results presented today, however, show that the clinical outcome is dismal. How can this be explained?

My associates and I have examined the matter by simulated aortic balloon dilations in a sophisticated circulatory model. We found that major pressure gradients appear only at critical stenoses and that these gradients are highly flow dependent. There is no gradient unless the aortic valve area drops to less than 0.7 cm^2, and if a gradient is present, it can be decreased very easily simply by lowering the cardiac output. The decrease in cardiac output will make a very substantial gradient virtually disappear. In reverse, these patients likely experience sudden return of the gradient if the cardiac output increases on exercise or if the ventricular function improves.

The most impressive finding, however, was that as the stenosis gets tighter and the flow becomes lower, the margin between success and death becomes more narrow. So, although it may be easy to stretch the aortic valve and improve the gradient either by enlarging the orifice or decreasing the output, if the valve becomes only a millimeter or two more stenotic, the good result not only disappears but the patient dies.

At the meeting of this Society 2 years ago, we told our cardiologist colleagues that balloon dilation of the calcified aortic valve will yield poor clinical results. Today, about 3,000 dilations and millions of dollars in expenses later, Dhillon and co-workers are telling them again through this carefully conducted study. I hope their warning will receive more attention.

I have two questions. First, there were 179 patients who had balloon valvuloplasty within a 2-year period. What happened to those who did not have operation?

Second, how on earth did you get 179 patients refusing surgical intervention in a 2-year interval?

DR DEVENDRA S. SAKSENA (Bombay, India): I congratulate Dr Dhillon and his group and agree with most of what they have said. At our institution in Bombay, my associates and I have a different set of patients, who are seen very late in the course of disease and are almost in New York Heart Association class V with cardiac cachexia. In our experience, cardiac cachexia is the single most important risk factor for morbidity and mortality in these sick patients. We perform balloon valvuloplasty primarily to improve the nutritional status of the patient and then within 3 to 4 weeks after a so-called successful balloon valvuloplasty, we go ahead and do a valve replacement. This has decreased our morbidity and mortality to levels acceptable to us. I think that is a good indication for the use of aortic balloon valvuloplasty.

It has been my observation that when I go back and operate on these patients within 3 to 4 weeks during the same hospitalization, the valve does not open at all. I do not know what has happened to reduce the gradient; maybe what Dr Robicsek said might be true. Dr Dhillon, when you operate on these patients after valvuloplasty, is the valve very open?

DR DHILLON: We agree that the term *valvuloplasty* is a misnomer. We recognize the excellent work that has been done by Dr Robicsek in the evaluation of the effect of balloon dilation.

Why were 179 valvuloplasties performed? The patients were referred to our institution from all over the United States. In fact, some of the referrals were from cardiac surgeons themselves. It is important to note that currently we do not recommend valvuloplasty for patients who want that procedure but can tolerate a valve operation.

The clinical results of valvuloplasty have been published by our institution. Roughly 25% of the patients will die, and clinical recurrence will develop in 25% within a matter of 6 to 9 months. We have had patients come back with restenosis who have again been refused surgical intervention because they are such poor candidates for operation. However, that has occurred in only a few patients.

We agree with Dr Saksena that this procedure offers really short-term palliation in a very select group of patients and that this can help these patients with their acute symptoms. The patients can then undergo valve operation, possibly with less risk, while the beneficial effects of valvuloplasty are still present.

Aortic Valve Debridement by Ultrasonic Surgical Aspirator: A Word of Caution

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Division of Cardio-Thoracic Surgery, Joseph B. Whitehead Department of Surgery, Emory University School of Medicine, Atlanta, Georgia

Aortic stenosis was relieved in 11 patients by ultrasonic debridement of the valve and annulus, while 102 other patients underwent valve replacement for aortic stenosis during 1988. Debridement was selectively applied based on findings of small annulus size (19 mm or less) and extensive calcification. Additional patient characteristics were mean transvalvular gradient of 78 mm Hg, advanced age, and marked left ventricular hypertrophy. Six patients had no residual gradient and 5 others a mean gradient less than 10 mm Hg. There were no complications related to the debridement process. Intraoperative transesophageal Doppler echocardiography demonstrated improved leaflet mobility and elimination of the gradient in all patients and elimination of associated valvular insufficiency in 2 patients. Follow-up echocardiography demonstrated late onset of new valvular regurgitation in 5 patients that was progressive and required reoperation in 3. Thickened, hardened, and retracted valve leaflets with loss of central coaptation were found in all 3 patients who underwent reoperation. Ultrasonic debridement can effectively relieve aortic stenosis, provide an excellent immediate hemodynamic result, and decrease operative time. However, the early occurrence of aortic insufficiency in a high percentage of patients makes it an unacceptable alternative to valve replacement, and the technique should be abandoned as a treatment for severe calcific aortic stenosis.

(Ann Thorac Surg 1990;49:746–53)

Mechanical debridement of the aortic valve was applied as treatment to relieve aortic stenosis for many years before successful valve substitutes were available [1, 2]. The recurrence of aortic stenosis in a high percentage of patients within 5 years as well as the emergence of successful prosthetic valve devices led to the abandonment of techniques of valve repair [3–5]. Recently, valve debridement using ultrasonic surgical aspiration devices has been reported as an effective alternative to valve replacement [6–8]. This report presents an experience using ultrasonic surgical debridement of the aortic valve in 11 patients to correct aortic stenosis and avoid valve replacement.

For editorial comment, see page 689.

Material and Methods

Eleven select patients out of a total of 113 patients undergoing operation for aortic stenosis underwent debridement of their aortic valve using ultrasonic surgical aspiration techniques. Nine of the 11 had severe aortic stenosis, with a mean gradient of 94 mm Hg (range, 80 to 128 mm Hg), and sclerotic trileaflet valves that were heavily calcified, with a small, calcified valve annulus (19 mm). Two patients were primarily undergoing coronary artery bypass grafting for ischemic symptoms but also had calcified aortic valves with moderate stenosis (mean gradient, 40 mm Hg; range, 36 to 45 mm Hg).

Intraoperative election to use the ultrasonic debridement technique was based on the desire to avoid implantation of a mechanical valve prosthesis in 9 elderly patients (mean age, 78 years) with severe aortic stenosis and a small calcified aortic root and annulus. Improvement in valve function without valve replacement was the indication in the 2 other patients with moderately stenotic valves who were primarily having coronary artery bypass grafting. Valve debridement as an alternative to valve replacement was discussed with all patients before operation.

Ultrasonic debridement was accomplished with a handheld probe that applied high-speed vibration at 23 kHz, a frequency above the human aural range, to the calcified atherosclerotic valve leaflets and annulus. Ultrahigh-frequency vibration impacts the harder elements of the sclerotic leaflet lesions and causes them to be dislodged from the softer tissues, which can absorb the vibration forces. Ultrasonic vibration fragments and disintegrates the calcific deposits, which are then aspirated from the remaining leaflet tissue. The ultrasonic vibrator probe was cooled by a continuous external irrigation of saline solution over the tip. Fragmented debris was aspirated from the operative field through the hollow core of the probe tip. Figure 1 demonstrates the debridement of a heavily calcified stenotic valve with resultant wide leaflet mobility and increase in orifice size. Debridement was performed using the Cavitron ultrasonic surgical aspirator (Cavitron Surgical Systems, Inc, Stamford, CT) in 9 patients and the Bovie ultrasonic surgical aspirator (MDT Castle, Inc, Rochester, NY) in 2 patients.

Presented at the Thirty-sixth Annual Meeting of the Southern Thoracic Surgical Association, Scottsdale, AZ, Nov 9–11, 1989.

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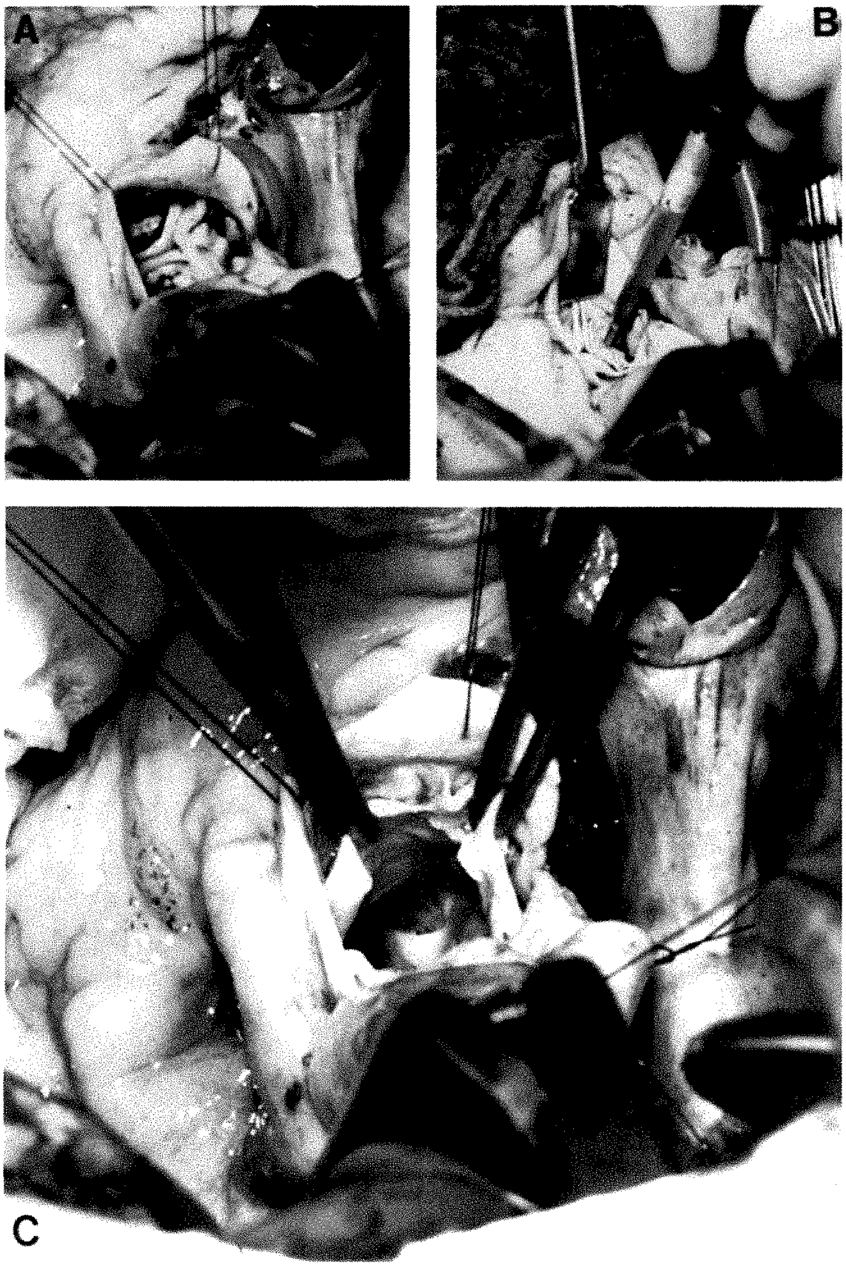

Fig 1. (A) Calcified aortic valve. (B) Debridement by ultrasonic technique in process. (C) Leaflets after completion of debridement process; note widely opened orifice area.

Effectiveness of the valve debridement was assessed by direct inspection and by simultaneous central aortic and intraventricular pressure measurements after the patient had been separated from cardiopulmonary bypass and hemodynamic stability had been obtained. Additional evaluation of leaflet mobility and the presence of any valve insufficiency was made by intraoperative transesophageal color flow Doppler echocardiographic comparison of preoperative and postoperative valve function and status. Later follow-up assessment was obtained by clinical examination and Doppler echocardiography, and by cardiac catheterization where needed.

In 3 other patients ultrasonic debridement was initiated but aborted when transleaflet calcification was found to be present and fenestration of a leaflet was produced (1 patient) or seemed likely to occur (2 patients). These 3 patients were treated with valve replacement and did well.

Anesthesia and techniques of cardiopulmonary bypass were identical to those employed for all patients undergoing aortic valve operations. Myocardial protection involved systemic hypothermia to 24°C and ischemic arrest with cold oxygenated crystalloid cardioplegia administered antegrade via the aortic root, coronary ostia, and/or coronary artery bypass grafts in 7 patients (mean, two grafts per patient).

Results

Operative Results and Follow-up

The transvalvular gradient was reduced from a mean of 94 mm Hg to a mean of less than 10 mm Hg (range, 0 to 20

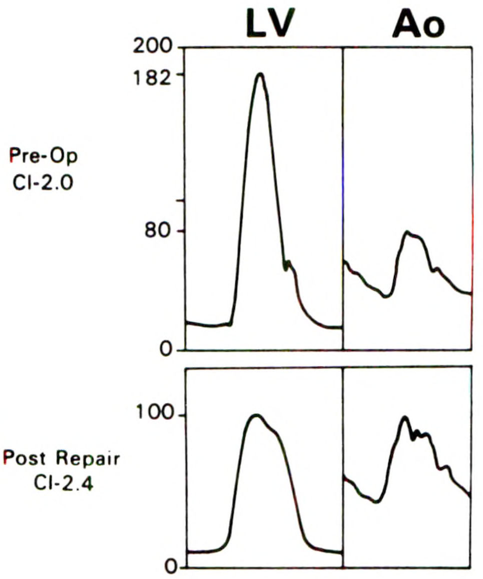

Fig 2. Aortic (Ao) and intraventricular (LV) pressure curves before and after valve debridement. (CI = cardiac index in liters per minute.)

mm Hg) in the 9 patients with calcified atherosclerotic trileaflet valves. In the 2 patients with moderately stenotic valves it was reduced from a mean of 40 mm Hg to a mean of 4 mm Hg (range, 0 to 7 mm Hg). Figure 2 presents aortic and ventricular pressure curves for a patient treated with ultrasonic debridement, demonstrating the effectiveness of ultrasonic debridement in eliminating the gradients associated with valvular stenosis. Intraoperative transesophageal color flow Doppler echocardiography demonstrated improved leaflet motion and freedom from serious valvular insufficiency in all patients. Two patients who had moderate aortic insufficiency preoperatively due to failure of effective leaflet coaptation were shown to have no insufficiency on the postoperative study.

All patients survived the operative procedure and did well hemodynamically. One patient died during the initial hospitalization of complications arising from a postoperative colon perforation. A Doppler echocardiographic study of her debrided valve before her death showed no gradient and no insufficiency. Ten patients were discharged and available for further follow-up.

Follow-up of the 2 patients with moderately stenotic valves after discharge has demonstrated improved leaflet motion and freedom from insufficiency. They continue to do well 2 years postoperatively.

Follow-up data on the 8 discharged patients who had severely stenotic trileaflet atherosclerotic valves treated by ultrasonic debridement reveals that 3 patients have re-

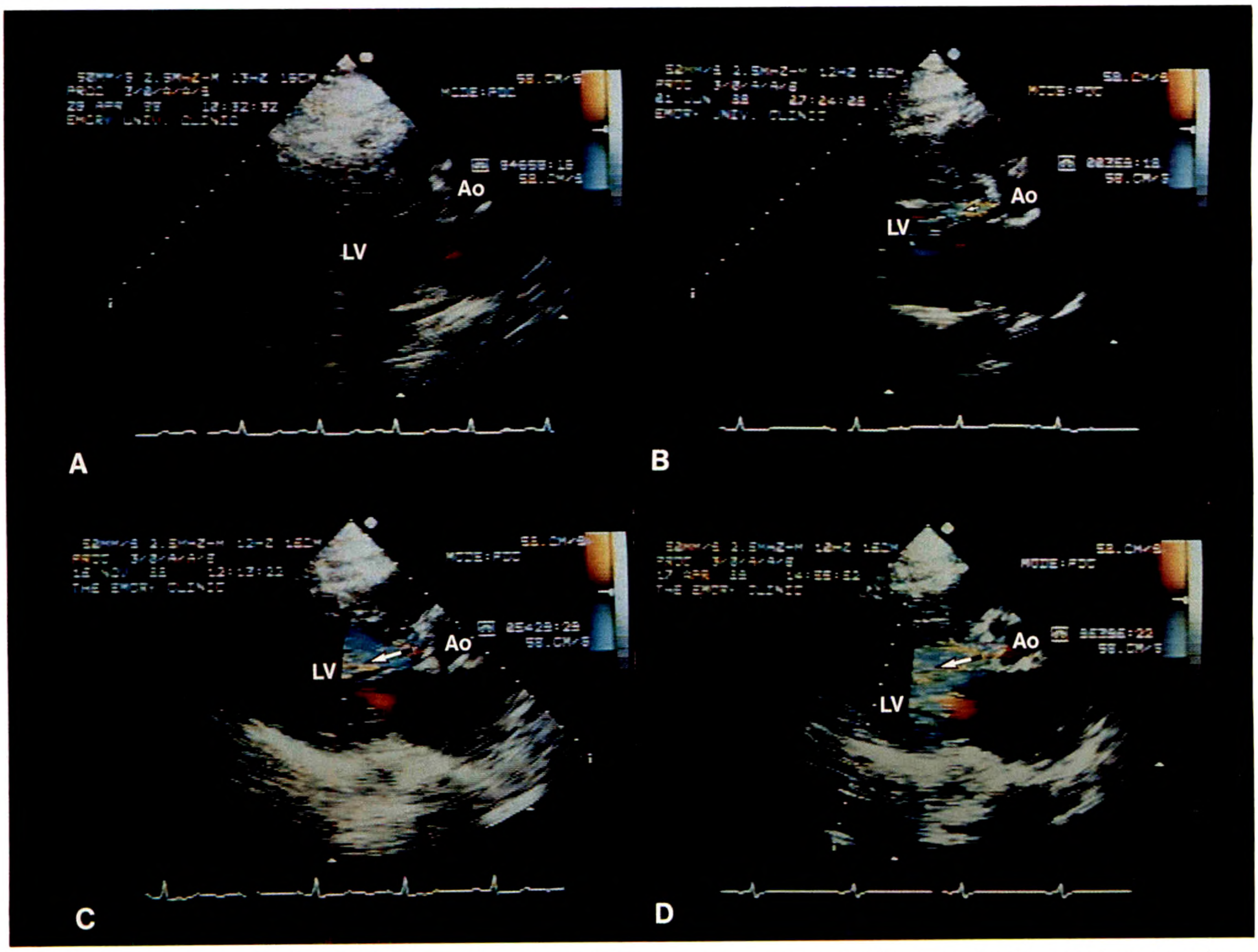

Fig 3. Serial postoperative color flow Doppler echocardiographic study (parasternal-long axis view) of debrided aortic valve showing progressive aortic insufficiency: (A) 1 week, (B) 6 weeks, (C) 7 months, and (D) 1 year. (Ao = aorta; LV = left ventricle.)

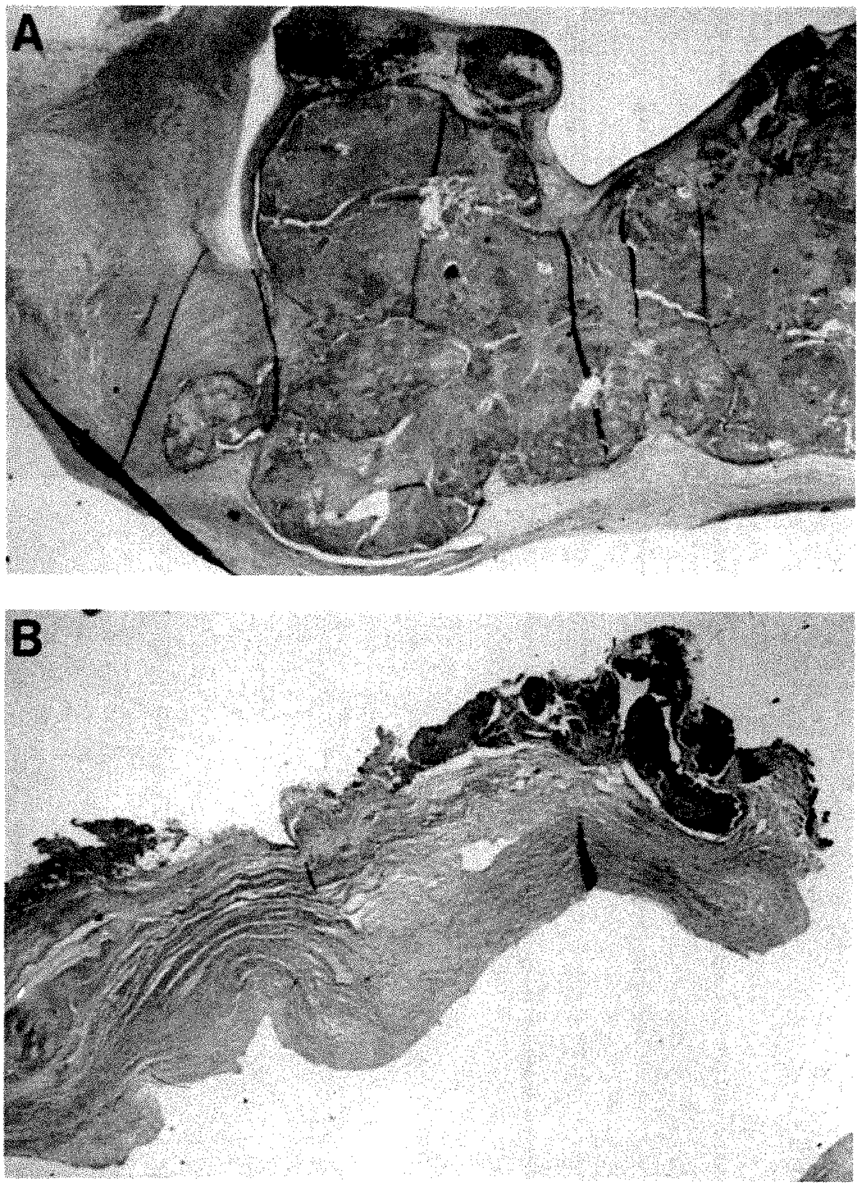

Fig 4. (A) Section of undebrided aortic valve leaflet showing pan-leaflet degeneration process of sclerosis myxoid degeneration and dystrophic calcification. (B) Section of partially debrided leaflet showing similar histology, but reduction in thickness.

quired reoperation and aortic valve replacement for progressive valve insufficiency. Two patients, aged 65 and 77 years, manage a quiet ambulatory existence without symptoms at daily activity but with mild valvular insufficiency documented at 19 and 13 months postoperatively. The 3 remaining patients (mean age, 80 years), all with chronic congestive heart failure preoperatively, have died 5 to 14 months postoperatively due to chronic respiratory insufficiency (1 patient) or sudden death (2 patients).

The 3 patients who have required reoperation and valve replacement were documented to have progressive aortic insufficiency on serial follow-up echocardiographic studies (Fig 3). They also exhibited failure to improve clinically, persistent or enlarging cardiomegaly on chest roentgenogram, and the presence of a new and increasing murmur of aortic insufficiency. Cardiac catheterization documented severe aortic insufficiency in all with mild or no aortic stenosis (maximum peak gradient, less than 30 mm Hg). Valve replacement required a 19-mm mechanical bileaflet prosthesis in each patient after further maximum annular debridement. Aortic root enlargement procedures were not felt advisable due to extensive annular calcification, age of the patients, and magnitude of the procedures. All patients did well, were discharged on warfarin anticoagulation, and remain clinically improved.

Histopathological Observations

The valve leaflets of several patients were available for study. Figure 4 presents histological sections of aortic leaflets from a patient in whom debridement was performed but had to be aborted due to fenestration. A nondebrided leaflet (Fig 4A) demonstrates histologically that the atherosclerotic calcified lesion of senescent aortic stenosis is a pan-leaflet process producing a thickened

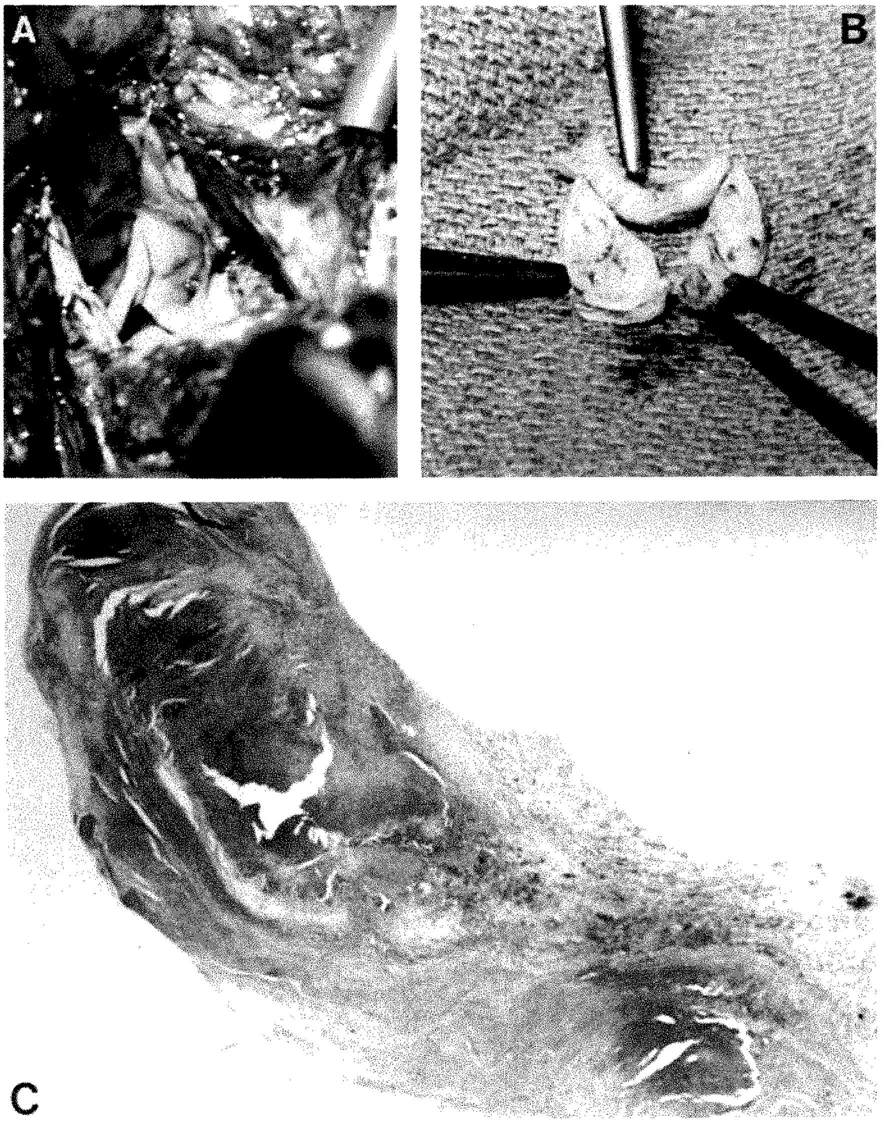

Fig 5. (A) Aortic valve with progressive insufficiency 1 year after ultrasonic debridement. (B) Excised thickened, shrunken, and retracted leaflets. (C) Histological section of excised leaflet showing marked fibrosis with sclerosis.

and sclerotic leaflet with focal areas of myxoid degeneration and extensive deposition of calcium within the fibrosed areas. It is intrinsic degeneration of the leaflet substance rather than a deposition on the leaflet surface. No normal leaflet anatomy is observed except the thin endothelial cell surface layers. Figure 4B shows a pathological section of a partially debrided leaflet that is histologically similar to the nondebrided leaflet. Sclerosis and calcification with focal myxoid degeneration is evident although the leaflet is thinner as a result of removal of one endothelial surface and central leaflet substances.

Figure 5A is an operative view of a valve in a patient with progressive valvular insufficiency who required reoperation and valve replacement 1 year after ultrasonic debridement. Grossly, the valve leaflets appear thickened, hardened, shrunken, and retracted (Fig 5B). Microscopic study of this leaflet tissue shows marked fibrosis with sclerosis and calcification. Neovascularization is prominent in areas but there is minimal myxoid degeneration (Fig 5C).

Comment

Early surgical procedures to correct symptomatic aortic stenosis were direct efforts to regain orifice area and improve leaflet mobility and thereby reduce the gradient without inducing regurgitation [9–11]. Before the advent of cardiopulmonary bypass and satisfactory valve substitutes, closed methods were used in the early 1950s by pioneering surgeons to relieve this critical lesion. Because of the rapid deterioration seen in patients with aortic stenosis once atrial fibrillation was added to the symptom triad of angina, syncope, or left ventricular failure, Harken and associates [9] advocated valvotomy by digital manipulation of the stenotic valve by way of an "operating tunnel graft" sewn to the ascending aorta. Muller and Hyman [10] and others reported some success using a transventricular mechanical dilator. These early efforts proved to be of limited success and associated with discouragingly high risks.

The limitations of these "blind" techniques led Mulder and Winfield [1] to advocate direct-vision commissurotomy using systemic hypothermia and three to nine minutes of aortic occlusion. However, they observed that "anatomic opening of the commissure is readily accompanied with some increase in valve orifice, but the heavy calcific deposits allow no improved valvular mobility." (This finding was recently repeated and confirmed by interventional cardiology colleagues using percutaneous aortic balloon valvoplasty.) This observation led Mulder and Winfield to advocate both direct commissurotomy and mechanical debridement of the calcified encrusted material from the leaflets under direct vision employing extracorporeal circulation and anoxic arrest. Three cases were presented in their original report in 1960 in which the "gradient was completely abolished and dramatic clinical improvement achieved." This led to a wide application of mechanical debridement for calcified aortic stenosis. Even though this experience took place in the infancy years of cardiac surgery and extracorporeal circulation, patient clinical improvement was clearly substantial and operative risks, high by current standards, were felt to be acceptable at the time given the high mortality of symptomatic aortic stenosis.

Unfortunately, the initial beneficial results were not always found to be lasting [2, 3, 12]. Reports [2, 5] demonstrated that restenosis occurs in the majority of patients within 5 years although clinical improvement was observed to be longer lasting in a minority of patients. Scannell and Austen [4] reported in 1966 that mechanical debridement compared favorably with aortic valve replacement at 3 years but not after 5 years. These and other reports of the improved and lasting results using new prosthetic valve substitutes led to the abandonment of mechanical debridement for the treatment of aortic stenosis.

Mindich and associates [13] reintroduced mechanical aortic valvoplasty in 1986 as an alternative to prosthetic valve implantation in select patients. Their initial report consisted of 23 patients with an average age of 75 years, all with acquired aortic stenosis, small aortic roots, and relative contraindications to anticoagulation. Valvoplasty was felt to be successful and an effective alternative to valve replacement if an orifice area greater than 1.1 cm could be achieved without producing more than mild residual aortic insufficiency. Only 2 patients required reoperation within 6 months to 6 years of follow-up: 1 for endocarditis and 1 for late valve "restenosis." Two patients died of unrelated diseases and 19 remained asymptomatic and free of thromboembolic complications; none required anticoagulation.

In 1988 Mindich and co-workers [6] expanded their valvoplasty techniques to utilize ultrasonic surgical aspirator debridement to decalcify stenotic aortic valves. Fifteen patients were reported who underwent aortic valve decalcification by the same methods and using the same equipment as in the present report. Although the characteristics of their patients were not defined specifically, our current population resembled very closely the elderly group with high gradients and small aortic roots that Mindich and co-workers reported in their 1986 series [13] having mechanical debridement. Their 1988 data on ultrasonic debridement related that the preoperative gradient in 35 patients was 47.5 ± 25 mm Hg, which was about one half that observed preoperatively in our series. Gradient reduction to 6.4 + 4 mm Hg and a significant increase in valve area to 1.6 ± 0.3 cm with no increase in insufficiency by Doppler echocardiography were similar to the immediate postoperative data for our series. No follow-up data beyond 6 months were given in an additional 1989 report by Mindich and co-workers [6, 8]. However, they advocated ultrasonic debridement as a possible safe and effective new treatment for aortic stenosis. They believed that it could be considered instead of valve replacement whenever severe valvular deformity or excessive insufficiency were not present.

Ultrasonic debridement of the stenotic aortic valve had been the subject of an earlier article by Brown and Davis in 1972 [14]. They modified the power and frequency of vibration of a dental device and demonstrated that it could effectively remove calcified material from excised aortic valve leaflet tissues. Two elderly patients were subsequently successfully treated by debridement of their stenotic aortic valves using the ultrasonic device. Both patients did well with excellent clinical early results, but only a 2-month follow-up was available in the initial report.

Freeman and colleagues [7] reported 15 patients with an average age of 78 years who had aortic valve decalcification for senescent aortic valve stenosis by ultrasonic surgical aspiration. Their evaluation again documented the effectiveness of the technique in relieving the gradient and improving leaflet mobility and coaptation. Their early data were very similar to those of the present series. However, no late follow-up data were available.

Without question ultrasonic surgical aspiration can debride calcified valve cusps and relieve aortic stenosis. However, the occurrence of regurgitation in 5 of 10 patients, including 3 patients with progressive regurgitation requiring reoperation and valve replacement, in the present series make its use most questionable. This is particularly of concern because the pathological mechanism of the insufficiency was seen to be inflammatory thickening and retraction of the debrided leaflets. An accompanying aortitis observed in 1 of the 3 patients makes one postulate that the ultrasonic technique elicits an intense "healing" process in the thin, flexible debrided leaflet that results in leaflet thickening and shrinkage as any other soft tissue wound would experience. It may be that the 2 patients with moderate aortic stenosis who have had a good result after valve debridement in this series have gained their "good" results from the fact that they needed commissurotomy and less extensive debridement. After removal of calcified deposits in these less diseased valves, the subsequent inflammatory reaction may have been less intense.

Until further data are available regarding late results of the current technique of ultrasonic debridement or until adjunctive measures are provided to eliminate the late occurrence of progressive insufficiency due to leaflet shrinkage, this technique should be held in reserve. This is unfortunate because it is a procedure that, once learned, can quickly debride fibrous, calcified leaflets responsible for aortic stenosis with an excellent initial hemodynamic result. If the occurrence of progressive postoperative insufficiency could be eliminated, it could be very useful in dealing with critically ill patients with severe aortic stenosis, small aortic roots, and contraindications to anticoagulation.

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DISCUSSION

DR THOMAS A. ORSZULAK (Rochester, MN): I think this is an important piece of information in the puzzle of aortic valve decalcification. In terms of this report, we could criticize its size and its narrowness but should recognize that with your critical selectivity in choosing patients in hopes of having it succeed, it still failed. If the results were superb we could at least say that ultrasonic debridement would work some of the time. It did not.

We have since had a further follow-up (beyond 9 months) for patients that you mentioned in your talk in whom we performed decalcification. We had a total of 61 patients. Twenty-six percent had severe aortic insufficiency at late follow-up, 30% had moderate insufficiency, and 14% required reoperation. Our original procedure was done with a mortality rate of 8.8%, and we have echoed your feelings in terms of abandoning the procedure.

I have three questions, and I have one challenge for you in terms of the report. Can you identify a line between too much and too little decalcification and perhaps find some niche for this procedure for aortic stenosis? Is the microscopic pathology perhaps a burn from the ultrasonic tip rather than just a healing process? And could platelet deposition and thromboxane interaction have anything to do with this scarring phenomenon?

The challenge is to address your comment in the report regarding mechanical valves being used in small roots due to the magnitude of the root-enlargement procedure, age of the patient, and extensive annular calcification. We have found pericardial patch enlargement of the aortic root always allows at least one, and rarely two, size increases in prostheses. With modest annular debridement of calcium, which may be facilitated with an ultrasonic surgical aspirator, closure of the aortotomy is enhanced with autologous pericardium with no aneurysm formation at 20-year follow-up. Our elderly population has a very high risk with the addition of Coumadin, and annular debridement enhances our choice of tissue valves.

DR STUART H. HARRIS JR (Lynchburg, VA): I enjoyed this report, particularly because I was privileged to be a resident with Dr Harken at Brigham in 1958 and 1960 and watched him do the finger valvoplasty. I then went down to Charlottesville and assisted Dr Muller using his transventricular valvulotome. I saw these procedures work, and I think it is a real tribute to these pioneers that they did. I also saw both of them open up the aorta, particularly after Dr Harken had obtained his pump, and do manual debridement of the aortic valve. I have also seen the valve tear when there was not an adequate valve to replace it. It was like being in an airplane when the motors went out and there was no parachute. I do think it is a tribute that in those days it did work at times. It is very interesting to see the nice advancement your report represents.

DR JOHN L. OCHSNER (New Orleans, LA): I think this is an important report and I am only sorry that it was not presented 3 years ago, because at the Ochsner Clinic we have operated on 18 patients from May 1987 to October 1988. Two of the patients died, late deaths unrelated to the procedure, but only 4 patients are without evidence of serious aortic insufficiency.

Aortic insufficiency, as you know, is seen in varying degrees and is not predictable. I think it is probably related to the amount of redundancy that one has in the free coapting edges of the leaflet when one completes the debridement. It is very analogous to the old fascia lata valves we did some 25 years ago. We found out that if we made the valve a little too big, and the coapting edges had somewhat of a serpentine configuration and were redundant, then it took a long time for the scarring and retraction to occur to the point where it became insufficient. I believe we obtained better results in those valves in which we had a lot of valvular tissue left; however, all of them will eventually contract to the point of insufficiency. I have to agree with Dr Craver that the procedure should be abandoned except for the use of the apparatus to debride the valve to put in a mechanical bioprosthesis or homograft.

DR HENDRICK B. BARNER (St. Louis, MO): My associates, Andrew Fiore and Larry McBride, have reported our experience with ultrasonic decalcification of the aortic valve to the Western Thoracic Surgical Association.

In 1988, we performed ultrasonic decalcification on 22 patients with a valve area of less than 0.8 cm^2 (mean, 0.7 cm^2), which increased to 1.42 cm^2 1 week postoperatively and was 1.29 cm^2 6 months later. The peak valve gradient decreased from 74 to 25 mm Hg and was 31 mm Hg 6 months postoperatively. Of concern to us has been the occurrence of aortic insufficiency, which was present in 50% of patients preoperatively and 87% of patients at 6 months. Four patients had new onset of aortic insufficiency and 3 patients had worsening of their aortic insufficiency, but no patient had more than 2+ regurgitation on a scale of 0 to 4.

Since analyzing these data, we have greatly curtailed our use of ultrasonic decalcification, but we have not abandoned the procedure totally. We believe that results are operator and patient dependent. At this point, we do not have enough data to analyze the influence of these variables on outcome, but we hope to do so by continued echocardiographic follow-up of our patients.

DR CRAVER: Dr Barner, we will be interested in the continuing follow-up of your patients. We will see if you can figure out some better method of patient selection or an adjuvant treatment for these debrided leaflets that would make the technique successful and the result long-lasting. Certainly it is a very gratifying procedure that relieves aortic stenosis. We just do not have any way to stabilize the leaflet membrane once it has been debrided and no model to take to the laboratory of the particular diseased tissue. If you could either treat the valve, increase the cross-links, or in some way keep the valve from shrinking, much as you treat pericardium with glutaraldehyde, it might well work.

Dr Ochsner, I appreciate your comments and would agree with what you are saying. In general, the amount of coaptation surface is the key to valve competency. And as you can see, in these senescent valves there seems to be quite a good area, usually 2 to 4 mm of leaflet coaptation, without annular dilatation. The insufficiency that we observed postoperatively is primarily a matter of progressive leaflet shrinkage, which produces inadequate leaflet coaptation rather than annular dilatation. This is supported by the elimination of aortic insufficiency in several patients who had it preoperatively after the valve leaflets were debrided and their coaptability restored.

Dr Harris, it was good to hear your comments. That must have been a fascinating time to live. I trained also with Dr Muller and I know that he is a man of great skill and bravery. To be a pioneer in cardiac surgery took great courage and persistence when the operative mortality in the early stages was 40%, because you knew that the patient mortality otherwise was 100%. And I have nothing but admiration for him and for the other pioneers who went through that era without parachutes that are employed today.

Dr Orszulak, thanks for bringing the experience from the Mayo Clinic. You certainly have had one of the widest experiences with this. I am sorry to see that your late insufficiency rate is also as high as the one we have observed. I do not think it is a matter of taking out too much of the disease process, because you are still left with the same underlying process. I think it is rather a matter of new scar formation, edema, and retraction. We looked carefully at those patients after the first one needed to be reoperated on to see if there was any indication of thickening of the leaflets by echocardiography, which may suggest a local thrombotic process or platelet deposition. We could see only the same degree of motion and leaflet thickness on the following echoes as we had before. So we still come back to scarring as the cause.

We have used root enlargement procedures for a decade and a half now and advocate those where needed. With this debridement technique, I had hoped to work out a way to expedite that process in very elderly patients. To debride the annulus, incise the noncoronary sinus, and insert a pericardial patch adds about 30 minutes to the operation, with some potential for bleeding. If the patients have any significant reason not to be able to take Coumadin, enlarging the aortic annulus and putting an appropriate sized porcine valve in is a valid alternative procedure. Because you can move up a from a size 19 easily to a 21 or 23, this gives the patient a quite good hemodynamic result.

Using the ultrasonic surgical aspirator just to debride the annulus is expensive. The disposable items that come with the Cavitron unit cost our hospital about $900 per case. If you start trying to reuse them multiple times, you get into problems of tip fracture in the device and burr formation on it. Also, I am just too frugal to spend that much money to do something I think can be done effectively with standard surgical methods.

Our overall mortality for aortic valve replacement in the group older than 75 years is 8.9%, taking all comers. So, I think using a root enlargement when you have to or a mechanical bileaflet valve when you cannot is probably preferable in managing patients with the very small aortic annulus with calcified aortic stenosis.

Donor-Specific Cytotoxicity Testing: An Advance in Detecting Pulmonary Allograft Rejection

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Ten beagle dogs underwent left unilateral lung transplantation and were treated with cyclosporine and azathioprine. After three days, cyclosporine administration was discontinued and azathioprine administration was maintained at a suboptimal dose to allow a graded response to the pulmonary allograft. Dogs were monitored by chest roentgenogram, bronchoalveolar lavage (BAL), and sampling of peripheral blood lymphocytes at weekly intervals. Open lung biopsies also were performed at weekly intervals in 5 of the dogs. Lymphocytes from the BAL and peripheral blood and those grown in culture from open and transbronchial biopsy specimens were tested for donor-specific cytotoxicity (DSC) by testing their responsiveness to lymphocytes from their donor. Rejection was confirmed in all animals. Donor-specific cytotoxicity became detectable in all animals in either the BAL fluid or biopsy specimens. An abnormal chest roentgenogram developed in 9 dogs, but not until after DSC was detected in the BAL in 7. In the open biopsy specimens tested for DSC, all exhibited DSC synchronous with the first changes noted on chest roentgenogram. Donor-specific cytotoxicity was sensitive for detecting rejection in 8 of 9 dogs in the BAL and in 5 of 5 dogs in the open biopsy specimens. Based on these results, 3 additional dogs were maintained on cyclosporine. Two of these dogs did not reject their transplants and had no evidence of DSC. We conclude that donor-specific functional assays provide an advance over less sensitive clinical techniques.

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Lung transplantation in its various forms is evolving as a therapeutic option for certain patients with end-stage pulmonary disease [1–3]. Current clinical methods of monitoring rejection, including chest roentgenogram, arterial blood gas, and pulmonary function tests, are sensitive but nonspecific in acute vascular and interstitial rejection. Clinical methods are also unreliable indicators for the more indolent form of obliterative bronchiolitis. Histological diagnosis by open lung biopsy and transbronchial biopsy is becoming the accepted standard. Fiberoptic bronchoscopy renders the pulmonary allograft more accessible than any solid organ allograft, and we have recognized that bronchoalveolar lavage (BAL) is useful in the early diagnosis of infection [4] and affords us the unique opportunity to sample sequentially a population of viable, immunocompetent cells from an entire lobe or segment of the pulmonary allograft. The analysis of cellular return permits the study of in situ graft immunity over time as well as monitoring of the response to treatment [5, 6].

The purpose of this study is to use our previously described model of pulmonary rejection in dogs [7] to compare chest roentgenogram, histology, phenotypic analyses, and functional studies of BAL-derived lymphocytes to predict early allograft rejection.

Material and Methods

To produce pulmonary rejection reliably and predictably, we performed left unilateral lung transplantation in 10 beagle dogs using a modification of the technique described by Veith and Richards [8]. Donor–recipient pairs were selected on the basis of responsiveness in the mixed lymphocyte cultures. The right lung in these dogs served as a control. The dogs were treated for three days with cyclosporine (15 mg/kg per day) and azathioprine (25 mg/day). After three days, when the animals had recovered from the operation, cyclosporine administration was discontinued and azathioprine administration was maintained to permit a graded response to the allograft. Three additional dogs were maintained on cyclosporine and azathioprine. At weekly intervals, all dogs underwent chest radiograph, BAL of left and right lungs, and sampling of peripheral blood lymphocytes (PBLs). In addition, 5 of these dogs underwent sequential open lung biopsy at weekly intervals.

Peripheral blood and BAL lymphocytes were expanded for four to seven days in the presence of tissue culture medium (TCM) consisting of RPMI 1640, 5% dog serum, 2 mmol/L glutamine, 25 mmol/L HEPES buffer, and 60 μg/mL gentamicin sulfate, supplemented with 50 U/mL human recombinant interleukin-2. The propagation of lymphocyte cultures from lung biopsy specimens was performed according to previously described methods for endomyocardial biopsy specimens [9]. Each fragment of biopsy specimen was divided into four to six pieces and cultured in a 96-well round-bottom plate (Costar, Cambridge, MA) with TCM and 50 U/mL interleukin-2 and 10^5

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irradiated (4,000 rad) autologous feeder cells. These feeder cells were prepared from PBLs by Ficoll Hypaque gradient centrifugation. Lymphocyte cultures propagated from BAL of both lungs, PBLs, and biopsy specimens were tested for cytolytic activity in a four-hour ^{51}Cr release assay using phytohemagglutinin-activated lymphoblasts of donor origin and third-party related controls. 10^6 target cells in 100 μL TCM were labeled with 100 μCi of $Na_2{}^{51}CrO_4$ (ICN Radiochemicals, Irvine, CA) for 90 minutes at 37°C and washed twice in TCM. 5×10^3 ^{51}Cr-labeled target cells in 100 μL of TCM were incubated with 100 μL of BAL, PBLs, or biopsy-propagated lymphocytes at an effector-to-target ratio of 10:1 or 40:1. After a four-hour incubation at 37°C, the plates were centrifuged, and 100 μL of supernatants were harvested and counted for radioactivity. Spontaneous chromium release was determined by incubating the target cells in TCM. Maximal release was determined by incubating the target cells with 5% Triton X. The percentage of cytotoxicity was calculated using the following formula (CML = cell-mediated lympholysis; Ex = experimental; M = maximal)

$$\%\text{CML} = \frac{\text{Ex cpm} - \text{Sp cpm}}{\text{M cpm} - \text{Sp cpm}}$$

(%CML >10% was considered as a positive test result.)

Samples of the BAL fluid from both lungs were sent for aerobic, anaerobic, and fungal cultures. Biopsy specimens were examined by conventional light microscope using standard hematoxylin and eosin staining. The following grading system was devised for the diagnosis of rejection: 1 = perivenular lymphocytic infiltration, 2 = periarterial and perivenular lymphocytic infiltration, 3 = perivascular and peribronchial lymphocytic infiltration, and 4 = elements of 1 through 3 with air-space involvement (Fig 1). All dogs were cared for in compliance with the "Guide for the Care and Use of Laboratory Animals" published by the National Institutes of Health (NIH publication No. 85-23, revised 1985).

Results

Thirteen of 18 dogs that underwent allotransplantation were used in this study. Failures included three operative deaths and two pulmonary infarcts secondary to pulmonary venous obstruction. All dogs were killed when their chest roentgenogram showed opacification of the allograft (13 to 34 days), and the tissue was examined histologically. One death occurred during the first follow-up secondary to an overdose of anesthetic. The following data reflect the results of the 10 dogs allowed to reject the transplant.

In 9 of 10 dogs, the roentgenogram showed predictable deterioration. The interval of deterioration varied from 13 to 34 days (mean, 17.8 days) (Fig 2). All animals were killed at the time deterioration was evident on roentgenogram, and the tissue was examined histologically. In every case, rejection was present, characterized by perivascular and peribronchial lymphocytic infiltration. In 8 of 10 dogs, advanced rejection (grade 3 or 4) was present at the time of autopsy, and 2 dogs showed minimal rejection (grade 1 or 2) (Fig 1).

Eight of 10 dogs exhibited DSC in the BAL with an interval of six to 22 days after transplantation (mean, 10.8 days). The donor-specific cytotoxic activity of lymphocyte cultures propagated from BAL of the lung allograft is shown in Table 1. This lytic activity was apparent as early as six to seven days after operation and in most dogs after 14 days. Bronchoalveolar lavage cells from the autologous control lung and PBLs did not exhibit cytotoxic activity. The CML response of allograft-derived cells was donor specific as no notable CML activity against third-party target cells was observed (Table 1). Five of seven BAL specimens and two of four biopsy cultures obtained from the transplanted lung exhibited donor-specific cytotoxic activity before radiographic changes (Table 2). Substantial CML reactivity was observed with BAL lymphocytes and biopsy-grown cells obtained from dogs with radiographic findings, indicating an ongoing process of rejection (Table 2). In every case in which CML activity was measurable, histology confirmed the presence of rejection (Table 3). Lymphocyte cultures obtained from two biopsy specimens in the first week after operation failed to exhibit CML activity, whereas biopsy-grown cells obtained eight or more days after transplantation exhibited substantial CML activity (Table 3). This donor-specific CML response of lymphocyte cultures propagated from BAL and lung allograft biopsy specimens was associated with a moderate to severe grade of rejection (Table 3).

In four of five biopsies performed sequentially, histological evidence of rejection preceded radiographic evidence of rejection by an interval of six to 16 days (mean, 9.7 days) (Fig 3). In three of the biopsy specimens, histological evidence of rejection occurred synchronously with appearance of DSC. In one of the specimens, the assay could not be performed for technical reasons. Another biopsy specimen showed no evidence of DSC, yet early rejection was present by histology.

In 2 of the 3 dogs maintained on immunosuppression, the roentgenogram never deteriorated; in 1 of these dogs, it showed a mild diffuse infiltrate 14 days after transplantation. Open lung biopsy was performed on this immunosuppressed group 14 days after transplantation and exhibited grade 1 histology in dogs with clear roentgenograms and grade 3 in the dog with the radiographic change. All these animals exhibited grade 1 or 2 histology at autopsy, and none exhibited DSC in the BAL. One dog did, however, exhibit DSC in biopsy-derived lymphocytes synchronous with radiographic and histological evidence of rejection.

Of the dogs that underwent transbronchial biopsy, the biopsy slightly underestimated the histology evident in open lung biopsy in 1 (grade 1 versus grade 2), another was an inadequate sample, and the last biopsy was in agreement with the histological findings of the open lung biopsy. Tests of donor-specific cytotoxic activity could not be performed on these specimens for technical reasons related to the small number of cells obtained with these specimens.

In all 10 dogs that were allowed to reject the transplant, aerobic, anaerobic, and fungal cultures either had no growth or did not grow a predominant organism. Of the dogs that were immunosuppressed, all 3 grew *Pseudomo-*

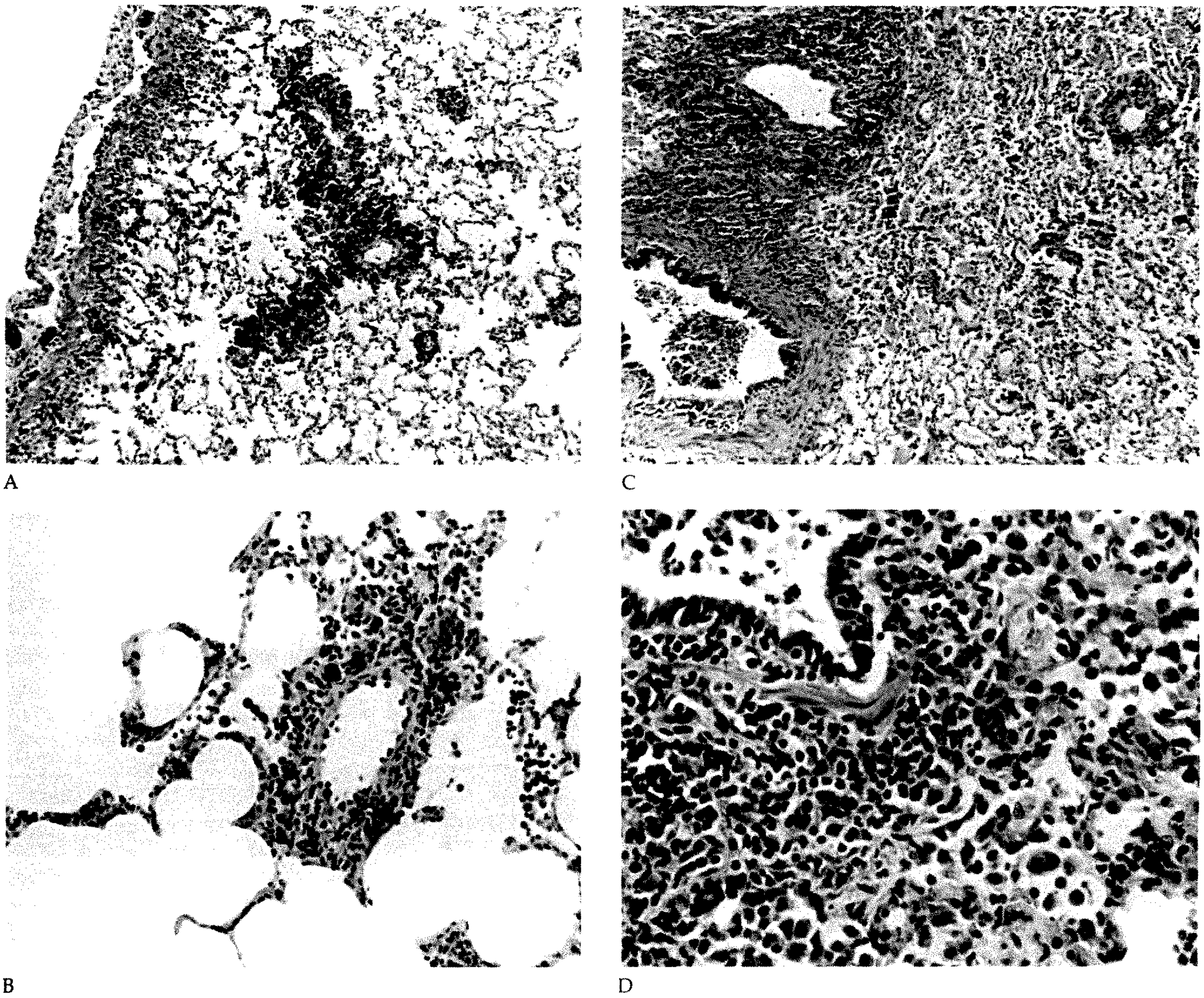

Fig 1. (A) Grade 1, acute lung rejection: a mild mononuclear inflammatory cell infiltrate cuffs the veins within the lobule, interlobular septa, and visceral pleura. (Hematoxylin and eosin, ×125 before 28% reduction.) (B) Grade 2, acute lung rejection: the mononuclear infiltrate surrounds the small veins and infiltrates beneath the endothelium, resulting in a hobnail configuration. (Hematoxylin and eosin, ×315 before 28% reduction.) (C) Grade 3, acute lung rejection: the inflammatory cell infiltrate encompasses the arteries (upper left) in addition to veins (upper right), while bronchioles are spared. Macrophages are prominent within the air spaces. (Hematoxylin and eosin, ×125 before 28% reduction.) (D) Grade 4, acute lung rejection: lymphocytes and immunoblasts infiltrate the walls of small bronchioles and intercolate between respiratory epithelial cells (upper left). Mononuclear cells extend into the alveolar septa and lie free within the air spaces. Occasional neutrophils are apparent. (Hematoxylin and eosin, ×500 before 28% reduction.)

nas aeruginosa on the 14-day follow-up. This was from the transplanted lung with a clear chest roentgenogram, normal cellular profile, and no clinical symptoms. All animals were treated, and subsequent cultures showed no growth.

Comment

Current methods of monitoring the pulmonary allograft do not appear to be sensitive enough to detect early rejection. In this study, radiographic deterioration was not apparent until moderate to severe rejection was present (grade 2 to 3). In the biopsy specimens of animals with clear chest roentgenograms, established early rejection was confirmed histologically (grade 1). Therefore, the chest roentgenogram was not a sensitive indicator of early rejection.

Functional studies of lymphocytes derived from the allograft enabled us to study the dynamics of the ongoing cellular events involved in the process of rejection. In previous reports, we showed evidence that BAL-derived lymphocytes exhibited donor-specific alloreactive T responses as assessed by increased proliferation exhibited in primed lymphocyte testing and DSC as measured in assays of CML [10, 11]. We have also shown that the accumulation of donor-specific cytotoxic cells is specific for the allograft as neither BAL from right lung controls nor PBLs showed CML activity. Norin and colleagues [12]

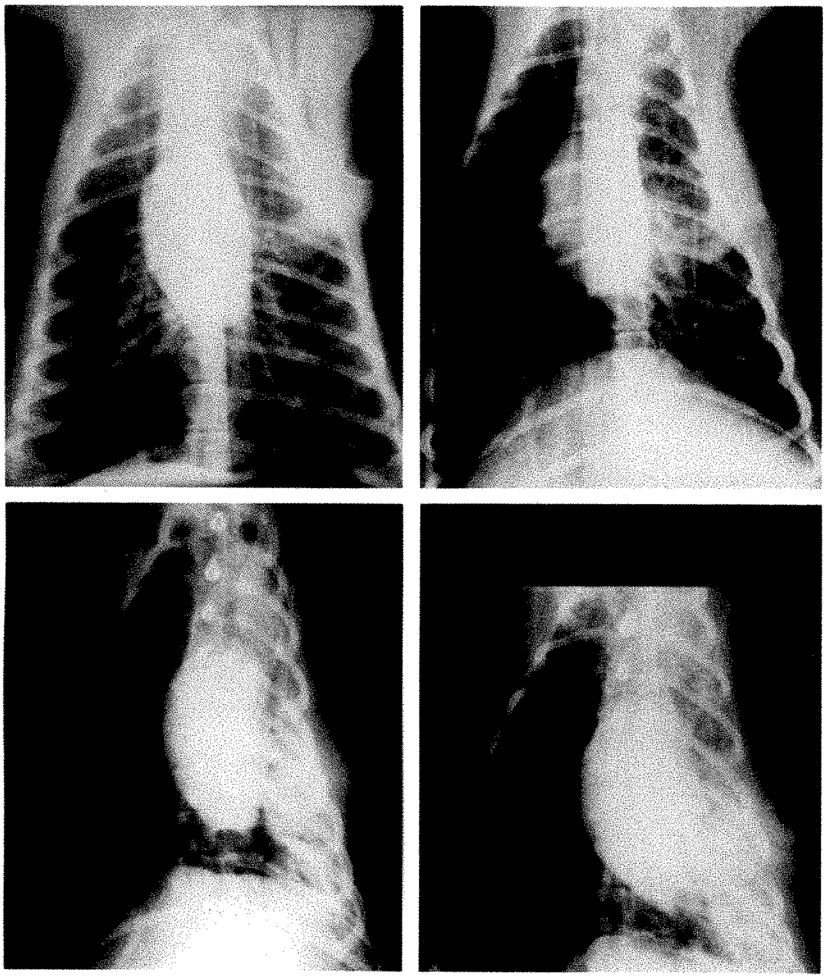

Fig 2. Radiographic progression of rejection at seven and 14 days (row 1, left to right) and at 21 and 28 days (row 2, left to right).

also demonstrated increased cytotoxic activity with BAL lymphocytes during rejection in the lung [12]. In this study, we focused on the presence of donor-specific cytotoxic cells in BAL and biopsy-derived lymphocytes in relationship with allograft status. Our results suggest that the presence of donor-specific cytotoxic cells in the BAL or biopsy specimens is an early predictor of rejection. In every case when DSC was measurable, early rejection was documented histopathologically. Also, cytotoxicity appeared quite frequently before radiographic evidence of a more advanced process. Although cytotoxicity was not

Table 1. Cytotoxicity of Bronchoalveolar Lavage and Peripheral Blood Lymphocytes of Canine Lung Allograft Recipients

Dog No.	Post-operative Day	Donor-Specific CML (%) BAL(L)	BAL(R)	PBL	Third-Party CML (BAL[L]) (%)
13	4	7	2	ND	ND
	11	2	2	4	0
	19	20	1	2	0
	27	24	4	3	1
3	6	21	2	ND	2
	13	19	ND	6	1
14	6	4	2	8	0
	13	23	1	3	0
8	7	76	5	3	1
	15	38	ND	ND	0
	22	31	4	ND	0

BAL = bronchoalveolar lavage; CML = cell-mediated lympholysis; L = left lung; ND = not done; PBL = peripheral blood lymphocytes; R = right lung.

Table 2. Cytotoxicity Versus Radiographic Findings

Radiographic Findings	Days After Operation	Donor-Specific CML (%) BAL	LBx
Clear	0–7	5, 21, 76	5
	8–14	2, 2, 23, 18	3, 67, 45
	15–21	20	. . .
Infiltrate	0–7	7, 20	. . .
	8–14	. . .	. . .
	15–21	24	. . .
Opacified	0–7	. . .	. . .
	8–14	19	37
	15–21	38, 31	31, 21, 41, 31

BAL = bronchoalveolar lavage; CML = cell-mediated lympholysis; LBx = lung biopsy.

Table 3. Cytotoxicity Versus Histology

Histological Grade of Rejection	Days After Operation	Donor-Specific CML (%)	
		BAL	LBx
Mild	0–7	2, 21, 21	5, 3
Moderate	8–14	20, 76	37, 67
	15–21	18	. . .
Severe	8–14	. . .	45
	15–21	38, 31	31, 21, 42, 31

BAL = bronchoalveolar lavage; CML = cell-mediated lympholysis; LBx = lung biopsy.

demonstrated in any of the immunosuppressed animals, their roentgenograms were normal, their biopsy results were consistent with only minimal degrees of allograft rejection, and their courses were benign.

These findings are similar to our experience with heart-lung transplant recipients in whom a significant association was found between the presence of alloreactive cells in BAL with pulmonary rejection [13]. Previous studies in our laboratory [14, unpublished observations] and by other investigators [15] have shown that donor-specific cytotoxic T lymphocytes propagated from endomyocardial biopsy specimens or the lung allograft are strongly associated with rejection of cardiac and lung allografts, respectively.

In summary, the chest roentgenogram is not an accurate predictor of early rejection. Fiberoptic bronchoscopy and the use of BAL renders the pulmonary allograft more accessible than any other solid organ allograft. By the use of BAL, we are able to repeatedly recover viable cells from a large area of the allograft in a minimally invasive manner and can study the immunologic events occurring in the allograft. We believe that this represents an advance over other less specific and less sensitive clinical techniques. Functional assays of BAL and biopsy-derived lymphocytes have been incorporated into our clinical program of heart-lung transplantation.

RADIOGRAPHIC FINDINGS

HISTOLOGY	CLEAR	INFILTRATE	OPACIFIED
MILD REJECTION	1	0	0
MODERATE REJECTION	3	2	1
SEVERE REJECTION	0	1	2

Fig 3. Histological grade of rejection present as compared with radiographic findings from the transplanted lung.

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Operation for N2 Small Cell Lung Carcinoma

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Of 48 patients with limited small cell lung carcinoma treated by different modes, but always including radical operation, a series of 25 patients with N2 lymph node metastases is reported. In a first period (1970 to 1977) treatment consisted solely of radical resection in 3 patients; chemotherapy was added to operation in 6, and local radiotherapy was added in 2. Since 1977, 14 patients were treated according to a comprehensive therapy protocol including radical resection (six pneumonectomies, one bilobectomy, seven lobectomies), chemotherapy, local radiotherapy, and prophylactic cranial irradiation. Eleven patients, in whom N2 disease was confirmed preoperatively, received chemotherapy as the first step, followed by "adjuvant" resection. Projected 5-year survival rate is 25% for the entire N2 group and 47% for the comprehensively treated group. Seven patients of this latter group are alive 12, 19, 30, 48, 66, 73, and 74 months after comprehensive therapy, equivalent to an observed 2-year survival rate of 38%. This is the largest reported series of patients with resected small cell lung carcinoma in the N2 stage treated at a single institution; the results are so encouraging that we can no longer advocate general refusal of radical lung resection for small cell lung carcinoma in the N2 stage if it is part of a multimodal therapeutic protocol.

(Ann Thorac Surg 1990;49:759–62)

In the 1970s, the opinion prevailed that small cell lung carcinoma (SCLC) no longer should be an object of surgical therapy. This attitude was founded on the then poor experience with operation as the sole treatment modality, the results of the British Medical Council study [1], and the surprisingly good initial response of these tumors to some of the newly available cytostatic agents. After the first enthusiasm for conservative treatment of SCLC had cooled, the publication by Shields and associates [2] triggered a new, more differentiated, and positive judgment of the surgeon's role in the treatment of early stages (I and II according to the TNM classification) of SCLC [3–9].

Concerning treatment of the more advanced stage IIIa, particularly in the case of N2 lymph node involvement, most investigators see no advantage in surgical intervention [10] and recommend only conservative therapy without citing stringent data [11, 12].

For editorial comment, see pages 691 and 692.

Other investigators [4, 8, 12–14] do indeed report on N2 patients who received surgical treatment, with some long-term survivors, but these few patients were not subjected to systematic analysis. Meyer and colleagues [12] reported in detail the treatment outcome of 10 patients with N2 metastases who had undergone a combination of chemotherapy, adjuvant resection, cerebral irradiation, and maintenance chemotherapy. The poor results (all patients died within 37 months) prompted Meyer and colleagues to recommend that operation not be performed in the treatment of these more progressive stages.

Recently, the Lung Cancer Study Group of the International Society of Chemotherapy published the outcome of 26 resected N2 cases treated postoperatively with two different treatment schedules of chemotherapy [15]; the value of this kind of treatment was considered uncertain.

In 1977 after a phase of diverse therapeutic approaches (1970 to 1977) at our institution, with close cooperation of surgeons, oncologists, and radiotherapists, a multimodal treatment scheme for limited SCLC was designed, including radical operation, chemotherapy, local radiotherapy, and prophylactic cerebral irradiation [7]; treatment is different for the early stages I and II and for stage IIIa. The experience with this multimodal therapy in N2 patients is reported.

Material and Methods

In the first phase (1970 to 1977), of 20 patients with limited SCLC, 11 with N2 involvement were treated with different modalities (Table 1). Since 1977, we treated 28 other patients according to the previously mentioned comprehensive protocol as follows:

Treatment A, for stages I and II
1. Radical surgery.
2. First cycle of chemotherapy.
3. First split course of local radiotherapy (25 to 30 Gy to the mediastinum and both supraclavicular regions).
4. Second cycle of chemotherapy.
   Restaging: If no tumor recurrence is detected:
5. Second split course of local radiotherapy (25 Gy) and simultaneously.

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Table 1. Treatment Details in 11 N2 Patients Noncomprehensively Treated

Treatment	No. of Patients
Resection only	3
Resection + chemotherapy (CAV)	6
Resection + radiotherapy	2

CAV = cyclophosphamide, adriamycin, and vincristine.

6. Prophylactic cerebral irradiation (30 Gy).

Treatment B, for stage IIIa:

1. First cycle of chemotherapy (for details, see Table 3). Precise radiological evaluation of therapeutic effect. In cases with at least a partial remission (minimum 50% reduction of all tumor manifestations), this is followed by:
2. Radical surgery in the extent of the original tumor spread.
3. Second cycle of chemotherapy.
4. First split course of local radiotherapy (25 to 30 Gy to the mediastinum and both supraclavicular regions).
5. Third cycle of chemotherapy.
   Restaging: If no tumor recurrence is detected:
6. Second split course of local radiotherapy (25 Gy) and simultaneously.
7. Prophylactic cerebral irradiation (30 Gy).

Corresponding to the rapid progress in chemotherapy during the last 12 years, the cytostatic regimen administered was changed; starting with cyclophosphamide, adriamycin, and vincristine [16] in the first years, we advanced to the protocol of Cohen and co-workers [17] (cyclophosphamide, adriamycin, vincristine, lomustine, methotrexate, and procarbazine), then to protocol 15/84 of the Schweizerische Arbeitsgemeinschat für klinische Krebsforschung, Bern, Switzerland (cisplatin, adriamycin, etoposide, cyclophosphamide, methotrexate, and vincristine), and, since 1986, to that of Evans and associates [18] (cyclophosphamide, adriamycin, vincristine, cisplatin, and etoposide). In all patients, accurate staging was the basis of all therapeutic decisions and histological diagnosis was confirmed by two independent pathologists.

Fourteen patients had histologically proven N2 involvement; clinical and treatment details of this subgroup are shown in Table 2. In 3 patients, N2 involvement was not diagnosed during pretherapeutic staging but was found only at histological examination of the resected specimen. Therapy for those patients was equal to that used for the early stages (treatment A).

Results

Figure 1 shows the treatment results of all 24 N2 cases and compares the Kaplan-Meier survival curves of the 14 patients with N2 metastases treated by the Innsbruck comprehensive therapy concept with that of 11 noncomprehensively treated patients who received only one or two therapy forms. A significant difference in the probability of survival was found between these two groups using the log-rank test ($p < 0.005$). Although all patients in the latter group died within 19 months, 3 of the 14 comprehensively treated patients are alive and free of tumor after more than 5 years (66, 73, and 74 months; median observation period, 20 months). Four patients of the comprehensive group either died before completion of therapy or refused one of the treatment modalities (see

Table 2. Treatment and Outcome for 14 Patients Treated Comprehensively

Patient No.	Sex	Age (yr)	TNM Stage	Operation	Chemotherapy	Local Radiation	PCI	Survival (mo)
Treatment B								
1	M	76	T1 N2 M0	Pneumonectomy	Evans[a]	– (Died)	– (Died)	7
2	M	63	T3 N2 M0	Pneumonectomy	Evans[a]	+	+	>12
3	F	43	T3 N2 M0	Pneumonectomy	Evans[a]	– (Died)	– (Died)	4
4	M	65	T2 N2 M0	Lobectomy	Evans[a]	+	+	>30
5	M	56	T2 N2 M0	Lobectomy	SAKK[b]	– (Relapsed)	– (Relapsed)	11
6	F	59	T1 N2 M0	Lobectomy	SAKK[b]	+	+	22
7	M	64	T1 N2 M0	Lobectomy	Cohen[c]	+	– (Refused)	32
8	M	43	T2 N2 M0	Pneumonectomy	ACO[d]/Cohen[c]	– (Relapsed)	+	9
9	M	57	T2 N2 M0	Lobectomy	Cohen[c]	– (Refused)	+	>66
10	M	59	T2 N2 M0	Bilobectomy	Cohen[c]	+	+	>73
11	F	59	T1 N2 M0	Lobectomy	Cohen[c]	+	+	>74
Treatment A								
12	F	66	T2 N2 M0	Lobectomy	Evans[a]	+	+	11
13	M	54	T2 N2 M0	Pneumonectomy	Evans[a]	+	+	>19
14	F	51	T3 N2 M0	Pneumonectomy	Cohen[c]	+	+	>48

[a] Reference 18. [b] Details of this protocol are available from the authors. [c] Reference 17. [d] Reference 16.

ACO = cyclophosphamide, adriamycin, and vincristine; PCI = prophylactic cerebral irradiation; SAKK = Schweizerische Arbeitsgemeinschat für klinische Krebsforschung.

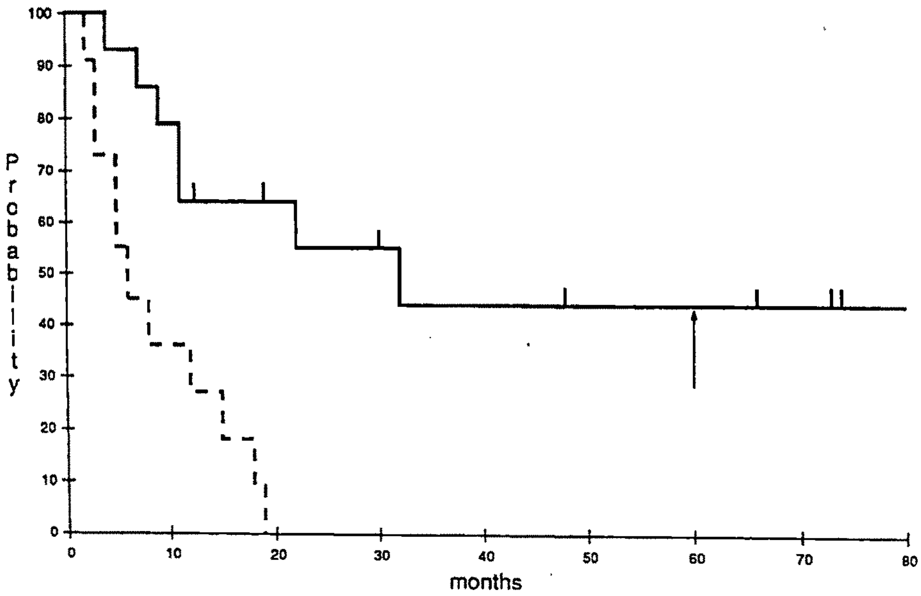

Fig 1. Kaplan-Meier estimate of survival of 25 patients with resected N2 small cell lung carcinoma. (Solid line, *14 comprehensively treated;* broken line, *11 noncomprehensively treated patients;* vertical bars *represent patients alive at last follow-up.)*

Table 2). One 42-year-old patient who underwent a left pneumonectomy after chemotherapy died of fulminant pneumonia of the right lung on the eighth postoperative day.

Six patients had tumor recurrence (Table 3). In a seventh patient a colonic carcinoma developed 42 months after onset of therapy for SCLC and was found to be inoperable.

Comment

The results show that long-term remission for more than 60 months in SCLC patients with mediastinal metastases can be achieved by intensive treatment including all possible therapeutic modalities. The theoretical considerations used by Meyer and colleagues [12] to explain their poor results do not appear to be valid. This is obviously true above all for the speculation that the initial chemotherapy might reduce the effectiveness of subsequent treatment.

Our results again raise the question of whether radical resection for N2 SCLC is appropriate within a comprehensive therapy concept or whether N2 lymph node metastases should be viewed principally as tantamount to incurability of the disease, meaning that operation is not indicated. Most investigators agree with the latter view without further debate, although no adequate data are available on this subject [13, 19]. The two most recently published articles in this field [10, 15] also give no clear answer: Karrer and Shields [15] find their results for patients with N2 disease inconclusive. In the discussion following the presentation of this paper, Shields underscored his opinion that a resection should never be performed in patients with N2 disease if the diagnosis has been established before operation and that pneumonectomy should never be considered for patients with SCLC. The Toronto Lung Oncology Group [10] also was not able to demonstrate a survival advantage for stage IIIa (N2) patients by adding operation to the conservative treatment modalities. Our data, however, indicate a benefit of additional operation in contrast to the results of these other studies.

Table 3. Site and Time of Relapse in 6 N2 Patients Comprehensively Treated

Localization	n	Survival (mo)
Locoregional	1	32
Locoregional and liver	1	9
Liver	2	11, 22
Brain	2	11, >18

An important argument in favor of resection in every operable stage of SCLC is the high incidence of regional relapses even after aggressive chemotherapy and radiotherapy [20–23], which are seen as the only relapse localizations until death. Whether such relapses are to be regarded as a possible source of secondary systemic metastases is not clear, but definitely should be considered. This would support the demand for a second, locally effective therapy, namely radical operation—including pneumonectomy—before use of regional radiotherapy.

N2 diagnosis generally is no contraindication for surgical therapy in non–small cell carcinoma. Because an SCLC diagnosis in itself does not pose an increased surgical risk, it makes no sense to exclude a patient from a therapy that with a different histology would be considered promising. Because long-term remissions of more than 60 months have not yet been documented for SCLC N2 patients after conventional treatment (chemotherapy and radiotherapy) but long-term survivors in this stage after multimodal treatment including operation have been reported in the present study and in other reports [4, 7, 13], the risk that patients will die of the disease if treated conventionally is evidently higher than the danger posed by operation.

The question of whether operation or chemotherapy should be the first therapy administered in the case of N2 metastases is still unanswered. An argument for using chemotherapy first is that after induction chemotherapy, more favorable safety zones are obtained at the resection lines, which without doubt basically must correspond to those required before therapy. When in the course of preliminary staging computed tomography and magnetic resonance imaging show no obstacles to resection, primary resection can be discussed when it is assumed, as for stages I and II [3], that the immediate removal of all recognizable tumor manifestations will create optimal oncological prerequisites for subsequent chemotherapy. This order is not part of our present treatment philosophy, but could be a subject for randomized studies in the future.

It is acutely necessary today that additional data be obtained on surgical therapy for SCLC in the N2 metastasis stage as this subgroup is the most numerous one among patients with limited disease.

We thank Dr Allan Gibbs of Cardiff, Wales, for reviewing the histological slides of our patients.

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Esophagogastrectomy Via Left Thoracophrenotomy

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Esophagogastrectomy is generally considered to be the treatment of choice for resectable tumors of the esophagus. Although many approaches and techniques have been advocated, since April 1983 we have used a left thoracophrenotomy approach for most lesions of the lower two thirds of the esophagus and gastric cardia. Stapling instruments have been used for mobilization of the stomach and fashioning of the esophagogastric anastomosis. One-hundred fifteen patients undergoing resection of malignant tumors with this technique were retrospectively reviewed. Perioperative mortality was 8.7% (10/115). The rate of anastomotic leakage was 1.7% (2/115), and benign narrowing of the anastomosis requiring dilation developed in 16 patients. The rate of recurrent anastomotic tumor was 4.3%. Eighteen patients had complications, and the mean postoperative hospital stay was 13 days. Survival at 3 years was 22.1%. During the period of study, 22 patients underwent esophageal resection by some other approach; the reasons for this are described. The advantages of the left thoracophrenotomy approach are discussed.

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Esophageal resection is currently accepted as the best form of treatment for operable tumors affecting the middle and lower thirds of the esophagus. Not only does it provide the best chance of curing the disease, but it is also the most effective method of palliation. The manner in which the operation is approached is controversial, however. Several different incisions, often in combination, are widely used. These include left thoracoabdominal [1]; laparotomy and right thoracotomy [2, 3]; three-stage resection comprising laparotomy, right thoracotomy, and a cervical incision [4]; the transhiatal approach [5, 6]; bilateral thoracotomies [7]; and left thoracophrenotomy, first described by Churchill and Sweet [8].

The left thoracophrenotomy approach to tumors of the middle and lower thirds of the esophagus and cardia of the stomach is still used by thoracic surgeons but has attracted little attention in the literature in recent years. We believe it offers advantages over other approaches and, since May 1983, have adopted it for routine resections of all esophageal tumors. This report describes our experience with this approach.

Material and Methods

The case records of all patients undergoing esophagogastrectomy under the care of one surgeon (R.J.D.) between April 1983 and February 1989 were retrospectively reviewed. During this period, a left thoracophrenotomy incision has been the preferred approach for this procedure and has been used in 115 patients. This group constitutes the major part of this report. Twenty-two patients have undergone esophageal resection by some other approach during this period. The various reasons for this are shown in Table 1.

Of the 115 patients who had a left thoracophrenotomy, 80 were men (69.5%) and 35 were women (30.5%). Their mean age was 63.2 years (±10.5 years; range, 33 to 84 years).

In addition to a full clinical history and examination, preoperative assessment consisted of barium swallow, biochemical and hematological screening, esophagoscopy, and, in the case of midthoracic lesions, bronchoscopy. Further investigations for evaluation of local or distant metastatic disease were performed only when indicated either clinically or from information derived from the routine preoperative assessment.

Histological cell types were as follows: adenocarcinoma, 60.8%; squamous, 35.7%; undifferentiated, 1.7%; small cell, 0.9%; and malignant lymphoma, 0.9%. Eighty-two tumors (71.3%) were located in the lower third of the esophagus and cardia, and 33 (28.7%) were in the middle third. No tumors in the upper third were resected by left thoracotomy. Staging was determined by histopathological examination of the surgical specimen.

A double-lumen endotracheal tube is used for anesthesia so that the left lung can be collapsed during operation. A left thoracotomy is made through the bed of the seventh rib. The skin incision extends forward almost to the costal margin, but the anterior costal margin itself is left intact. The esophagus is mobilized from the diaphragmatic hiatus to a point at least 5 cm above the upper level of the tumor. When we attempt a curative resection, we extend mobilization behind and above the aortic arch, taking care to preserve the recurrent laryngeal nerve. If necessary, the arch is mobilized by dividing one or two intercostal branches. We have not found it necessary to resect the seventh rib. The diaphragm is opened in line with the wound, leaving a 1-inch cuff anteriorly to facilitate closure.

The stomach is mobilized using the LDS stapler (Auto-

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Table 1. Incisions Other Than Left Thoracophrenotomy, 1983–1989

Incision and Reason	n
Left thoracoabdominal	
Tumor clearance	5
Previous operation	2
Poor exposure	2
Previous benign perforation	1
Acute perforation	1
Unspecified	2
Total	13
Cervical plus left thoracophrenotomy	
Small proximal thoracic esophagus	3
Proximal tumor	2
Total	5
Right thoracotomy plus laparotomy	
Tumor at level of arch/left main bronchus	2
Previous left thoracic operation	1
Suspicious nodule in right lung	1
Total	4
Total	22

suture, UK) around the greater curvature except for the short gastric vessels, for which the Surgiclip instrument is used. After division of the short gastric vessels, the gastrocolic omentum, the left gastric artery, and the lesser omentum, the stomach is delivered into the left hemithorax through the hiatus. It is transected using a linear stapler at a site at least 5 cm distal to the lower margin of the tumor. The esophagus is divided proximally, and the tumor-bearing segment is removed. More recently, however, we have temporarily left the posterior wall intact to facilitate introduction of the detachable anvil of the end-to-end anastomotic (EEA) stapling instrument, after which the remaining part of the esophagus is divided.

Esophagogastric continuity is normally restored using the EEA stapler (Autosuture) as previously described [9, 10]. The 25-mm head was used in 34 patients, the 28-mm head was used in 64, and the 31-mm head was used in 15. In 3 patients, initial sizing with the 28-mm head led to mucosal tearing of the proximal esophagus and it was necessary to use the 25-mm head to perform the anastomosis more proximally. In 2 patients, a hand-sewn anastomosis was necessary because the proximal esophagus was too small for the 25-mm head to be inserted. Eighty anastomoses (69.5%) were constructed below and 35 (30.5%) were constructed above the aortic arch. In this latter group, the stomach is routed lateral to the arch. After placement of a nasogastric tube, the anterior gastrotomy through which the EEA stapler was inserted is closed with a linear stapler. All straight staple lines are oversewn with a continuous 3-0 Mersilene suture. Neither fundoplication nor pyloroplasty are routinely used. Patients are generally extubated in the operating room and returned directly to the general ward.

After operation the nasogastric tube is left open to air until the drainage is minimal, at which time it is removed, generally on the second postoperative day. Crystalloids are administered through a peripheral vein for fluid maintenence, and patients are allowed to drink water on the fourth day. Oral intake is advanced to soft solids over the next three days. Contrast radiographic studies are obtained only if there is clinical evidence to suspect an anastomotic leak or if there was difficulty performing the anastomosis at the time of operation.

After discharge from the hospital, patients are seen at 3-month intervals and assessed for signs of tumor recurrence and anastomotic stricture formation. Patients with dysphagia undergo esophagoscopy and dilation if necessary. Follow-up to May 1989 was complete in 99.1% of patients.

Radiotherapy and chemotherapy were not used preoperatively in this series and postoperatively were used only for palliation of recurrent or residual disease. Survival was calculated according to the method described by Grunkemeier and Starr [11].

Results

The TNM status of the tumors as deduced from histological examination of the resected specimens showed that 37 tumors were stage 1, 7 were stage 2A, 70 were stage 3, and 1 was stage 4. The gastric and esophageal resection margins were free of tumor in 108 (93.9%) of the specimens.

Operative mortality, defined as deaths within 30 days or during initial hospital stay, was 8.7% (10/115). The cause of death was anastomotic leakage in 1 patient, pneumonia in 4, pulmonary embolism in 2, chylothorax in 1, septicemia in 1, and myocardial infarction in 1. Eighteen patients (15.6%) had postoperative complications, although these did not delay discharge from the hospital for 8 patients. Complications that could be attributed to operative technique included one anastomotic leak, one gastrotomy leak, and one chylothorax. Patients were discharged from the hospital at an average of 13.1 days (±10.5; range, six to 99 days) after operation.

Anastomotic leakage developed in 2 patients (1.7%), 1 of whom died as a result. The leakage was successfully repaired in the other patient three days after operation.

Narrowing of the anastomosis sufficient to cause dysphagia and relieved by dilation developed in 16 patients. In 13 of these, a single dilation was sufficient to restore normal swallowing. There was no statistically significant difference between the rates of benign stricture formation of the three sizes of anastomotic stapler. Malignant strictures at the anastomosis occurred in 5 patients (4.3%).

Survival is shown in Figure 1 and was 57.0% at 1 year, 32.3% at 2 years, and 22.1% at 3 years.

Comment

Controversy persists regarding the optimal surgical approach for esophagogastrectomy, and a variety of techniques have been advocated. These include left thoracophrenotomy [8, 12–14], right thoracotomy and laparotomy [2–4, 15], left thoracoabdominal [1, 16], left thoracotomy and midline laparotomy [17], bilateral thora-

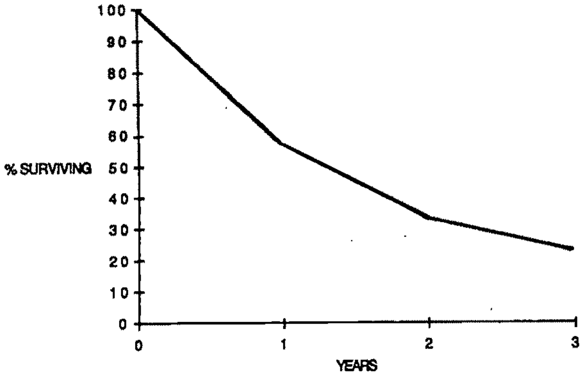

Fig 1. Actuarial 3-year survival of 115 patients undergoing esophagogastrectomy for malignant disease via left thoracophrenotomy.

cotomies [7], and laparotomy and cervical incision without thoracotomy [5, 6, 18]. A neck incision with cervical anastomosis may be added to any of these approaches.

None of these various methods have made any significant difference in long-term survival, although some investigators consider subtotal esophagectomy to be important in this regard. Alternative techniques have generally been recommended and found favor among different surgeons for their ease of performance of a difficult operation and the consequent reduction in morbidity and mortality that should result. The left thoracophrenotomy approach is historically important as the method advocated by Sweet and others in the pioneering days of esophageal surgery. Although this approach is still practiced by some thoracic surgeons on both sides of the Atlantic, little appears in the literature about this technique. We believe that it offers several advantages over other methods for tumors of the middle third of the esophagus and below; for this reason, we report our experience with the technique.

If the thoracotomy incision is made at the correct level through the sixth interspace, good access is available to the entire intrathoracic and abdominal esophagus, including the portion behind the aortic arch where dissection can be facilitated if necessary by mobilization of the aorta. Care must obviously be taken with the recurrent laryngeal nerve, and the course of the thoracic duct must be borne in mind. The supraaortic esophagus can be visualized and mobilized by dividing the pleura lateral to the left subclavian artery, and esophagogastric continuity can be reestablished by anastomosis at the apex of the thorax. We have not found it necessary to resect the seventh rib [14], but very rarely we make a second incision through the fourth interspace. This has been necessary only in patients requiring a thoracoabdominal incision.

The left thoracophrenotomy incision offers adequate exposure of the stomach and excellent access to the short and left gastric arteries through the opening in the left hemidiaphragm. Even performance of pyloroplasty is often possible, although we do not perform it routinely. Opening and closing of the wound is rapid and simple and, although we have not attempted to quantify them, it is not unreasonable to assume potential benefits in terms of reduced operating time and a smaller incision with consequent greater patient comfort and reduced wound morbidity.

Stapling instruments have proved to be very useful in performing esophagogastrectomy through the left side of the chest. Not only do they facilitate mobilization of the stomach through the diaphragm, but use of the EEA stapler allows rapid and reliable creation of the anastomosis [9, 10, 19–21] at the apex of the left thorax, so that it is nearly cervical in location. Proponents of the cervical anastomosis argue that, should a leak occur, extravasation is confined to the neck, causing less morbidity than an intrapleural leak. Our rate of anastomotic leakage is low, and we do not believe that there is sufficient justification for a separate neck incision with its consequent cosmetic effects and prolongation of operating time.

We have not found the left thoracophrenotomy approach to be associated with a significantly higher incidence of postoperative complications as compared with other approaches [22]. The rate of stricture formation was similar to that previously reported [23].

Some of the more frequently used alternatives to the left thoracophrenotomy approach are the right thoracotomy plus laparotomy or the left thoracoabdominal incision, either of which can be combined with a cervical incision, and the transhiatal esophagectomy. Arguments in favor of the right thoracotomy plus laparotomy approach are that a more complete tumor resection can be obtained and that a higher anastomosis can be created. We believe that an equally complete resection is possible through a left thoracotomy and that, particularly with use of the EEA stapler, an anastomosis at the apex of the chest is relatively straightforward. The left thoracoabdominal approach, as used by us before 1983, provides little added exposure over a left thoracophrenotomy and poses potential problems associated with division of the costal margin and anterior abdominal wall musculature. The transhiatal approach, as advocated by Orringer and associates [5, 6], has been criticized for violating the basic surgical principles of adequate exposure and hemostasis and for an excessive mortality and morbidity. Although Orringer and associates have argued these points well, Shahian and colleagues concluded [24], and we agree, that this approach should be viewed as an addition to the surgical armamentarium rather than a replacement of standard techniques.

During the course of this series, we have occasionally elected to use one of the alternative approaches for the reasons listed in Table 1. In addition, we have not hesitated during the course of operation either to extend the incision to the abdominal wall because of difficulties of access or tumor bulk or to perform the anastomosis in the neck if the proximal esophagus is too small or involved by tumor.

Our results with esophagogastrectomy by left thoracophrenotomy are comparable with those of published series using alternative methods. We believe that this technique has advantages of its own and is worthy of its place in modern thoracic surgery.

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Comparison of Prostacyclin Production of Human Gastroepiploic Artery and Saphenous Vein

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The prostacyclin production of the gastroepiploic artery (GEA) and saphenous vein (SV) were studied in 5 patients undergoing coronary artery revascularization. The GEA produced 90.0 ± 11.9, 132.4 ± 13.7, and 191.1 ± 21.8 pg/mg tissue (mean ± standard error of the mean) of 6-keto-prostaglandin $F_{1\alpha}$ (prostacyclin metabolite) after 2.5, 5.0, and 10.0 minutes, respectively, of incubation in Krebs–Henseleit buffer at 37°C. The SV produced 39.8 ± 7.0, 66.7 ± 9.1, and 123.6 ± 15.1 pg/mg tissue of 6-keto prostaglandin $F_{1\alpha}$ after 2.5, 5.0, and 10.0 minutes, respectively, of incubation. The GEA produced significantly more 6-keto-prostaglandin $F_{1\alpha}$ than SV at all three sampling times up to ten minutes of incubation ($p < 0.01$). Prostacyclin is a potent vasodilator and an inhibitor of platelet aggregation. Prostacyclin production by the internal mammary artery was reported to be much higher than that of SV, and the patency rate of internal mammary artery grafts is reported to be better than that of SV grafts in coronary artery revascularization. Therefore, our results suggest that the patency rate of GEA grafts may be better than that of SV grafts in coronary artery revascularization. The GEA is a promising and excellent graft from the biochemical point of view.

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The right gastroepiploic artery (GEA) seems to be a very promising arterial bypass graft for coronary artery revascularization. The saphenous vein (SV) has been widely used as bypass graft material; however, the late patency rate of the SV graft is clearly poorer than that of the internal mammary artery (IMA) graft [1]. Thus, the IMA graft is the first choice for revascularization of the key coronary artery. The IMA graft(s) and SV graft(s) are now a standard combination for coronary artery revascularization. Another arterial graft substitute is frequently required not only for complete arterial coronary revascularization, but also in many cases of coronary reoperation. The GEA is a good candidate artery from the anatomical point of view.

Historically, the GEA had been applied in myocardial implantation [2], just as the IMA graft was used by Vineberg [3]. Suma and associates [4], Pym and coworkers [5], and Lytle and colleagues [6] used GEA as an in situ graft for direct coronary anastomoses. Suma and associates [7] reported the early patency of GEA grafts as satisfactory. However, the late patency of the GEA is yet to be proved.

Chaikhouni and colleagues [8] described the biochemical explanation for the reported superior patency rate of the IMA graft over the SV graft. They showed that the endothelium of the IMA produced more prostaglandin I_2 (prostacyclin) than that of the SV. Prostacyclin is a potent vasodilator and an inhibitor of platelet aggregation [9].

In the present study we compared prostacyclin production of the GEA and the SV. The higher prostacyclin production by the GEA suggests that the GEA graft would show a higher patency rate than the SV graft.

Material and Methods

The GEA and SV grafts were harvested simultaneously from each of 5 patients undergoing coronary artery revascularization. The remnant segments of them were frozen immediately in liquid nitrogen and kept at −70°C until assayed. The GEA and SV were not dilated mechanically. The frozen remnant segments were soaked in Krebs-Henseleit bicarbonate buffer, cut into 0.5 to 1.0-cm sections, and weighed. Each piece of vessel was incubated in 3.0 mL of the medium at 37°C. The medium consisted of Krebs-Henseleit bicarbonate buffer (NaCl, 118 mmol/L; KCl, 5.4 mmol/L; $MgSO_4$, 1.0 mmol/L; $CaCl_2$, 2.5 mmol/L; Na_2HPO_4, 1.1 mmol/L; $NaHCO_3$, 25 mmol/L; *d*-glucose, 10 mmol/L), and was bubbled with mixed gas (O_2, 95%; CO_2, 5%). At 0, 2.5, 5.0, and 10.0 minutes of the incubation period, a 0.5-mL aliquot of the medium was taken into 20 mL of 15% C_2H_2OH with 200 μL of 1 N HCl.

After this medium was passed through a Sep-Pak C_{18} cartridge, 6-keto-prostaglandin $F_{1\alpha}$ (6-keto-$PGF_{1\alpha}$) was selected using reversed-phase high-performance liquid chromatography (Hitachi 655) C_{18} and dried. 6-Keto-prostaglandin $F_{1\alpha}$ is a stable hydrolysis product of prostacyclin and was measured by radioimmunoassay (6-keto-$PGF_{1\alpha}$ [^{125}I] RIA Kit, Dupont [10]).

The results were reported as picograms of 6-keto-$PGF_{1\alpha}$ per milligram of tissue for the incubation period, and the production of 6-keto-$PGF_{1\alpha}$ (in nanograms) was plotted against the wet weight of the vessel. The data were statistically analyzed by Student's *t* test to determine

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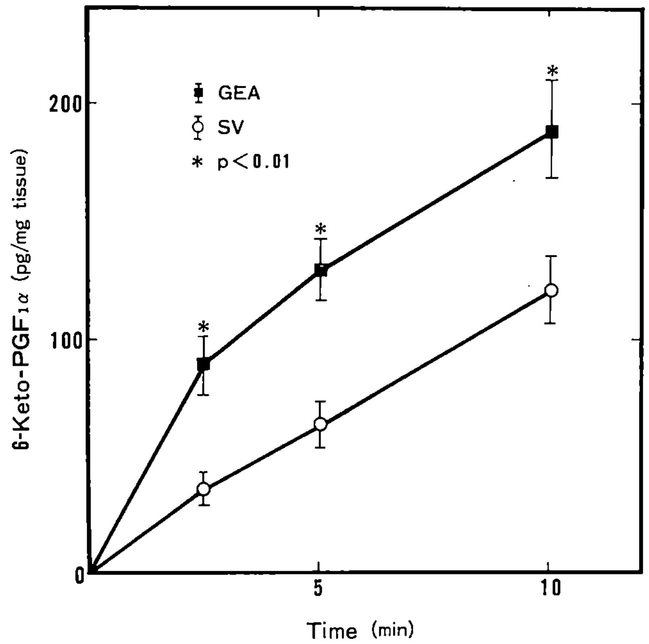

Fig 1. Gastroepiploic artery (GEA) produced significantly more 6-keto-prostaglandin $F_{1\alpha}$ than did saphenous vein (SV) at all three sampling times up to ten minutes of incubation (GEA, n = 5; SV, n = 5; mean ± standard error of the mean).

significant differences. Differences were considered significant at $p < 0.05$.

Results

The production of 6-keto-$PGF_{1\alpha}$ by GEA was 90.0 ± 11.9 pg/mg tissue (mean ± standard error of the mean), whereas that by SV was 39.8 ± 7.0 pg/mg tissue ($p < 0.01$) at 2.5 minutes of incubation. At 5.0 minutes, the production of 6-keto-$PGF_{1\alpha}$ by GEA was 132.4 ± 13.7 pg/mg tissue, whereas that by SV was 66.7 ± 9.1 pg/mg tissue ($p < 0.01$). At 10 minutes of incubation, the production of 6-keto-$PGF_{1\alpha}$ by GEA was 191.1 ± 21.8 pg/mg tissue, and that by SV was 123.6 ± 15.1 pg/mg tissue ($p < 0.01$). The GEA produced significantly more 6-keto-$PGF_{1\alpha}$ than SV at all three sampling times up to ten minutes of incubation (Fig 1).

The 6-keto-$PGF_{1\alpha}$ production of GEA and SV at ten minutes of incubation was plotted against the vessel weights (Fig 2). Production of 6-keto-$PGF_{1\alpha}$ by GEA and SV was positively linearly correlated with vessel wet weight.

Comment

Many authors have discussed possible factors that affect graft patency in coronary artery revascularization. These were divided into two groups. The first group attempts an explanation from a mechanical point of view, whereas the other group's explanation relies on biochemical factors. Mechanical factors include the host factors (condition of the native coronary arteries, left ventricular wall motion), mechanical characteristics of the graft, and the surgical technique [11, 12]. Biochemical factors included prostacyclin production by the graft material, plasma cholesterol, triglyceride, and glucose [8, 13, 14].

Prostacyclin is the major prostaglandin synthesized by the endothelial cells. A small amount of prostacyclin is also produced by fibroblasts and smooth muscle cells. Prostacyclin is a potent inhibitor of platelet aggregation and a vasodilator acting directly on the smooth muscle cells of the vessel [9] (Fig 3). The precursor of prostacyclin is arachidonic acid, which is converted from phospholipids by phospholipase. Arachidonic acid is first converted to prostaglandins G_2 and H_2, by cyclo-oxygenase and peroxidase, and then to prostacyclin under the influence of prostacyclin synthetase. Prostacyclin is rather unstable and largely degraded spontaneously to 6-keto-$PGF_{1\alpha}$, with a half-life of only a few minutes in whole blood. 6-Keto-prostaglandin $F_{1\alpha}$ is biologically inert [15]. Prostacyclin binds to a surface receptor on platelets and stimulates production of cyclic adenosine monophosphate, which is an inactivator of platelets. Cyclic adenosine monophosphate prevents shape changes of platelets for secretory function and inhibits receptor-mediated binding to platelet surface of factor VIII/vW and fibrinogen [16]. Higher productivity of prostacyclin by graft surfaces may induce less thrombosis, more vasodilatation, and ultimately a better patency rate of the bypass grafts.

In the present study, we measured the 6-keto-$PGF_{1\alpha}$ production of the GEA and the SV using radioimmunoassay, which is currently the most established and accurate method available [17]. Figure 4 shows the accuracy of our measurement technique. The 6-keto-$PGF_{1\alpha}$ production from the GEA was more than that of the SV, regardless of time or the weight of the vessels.

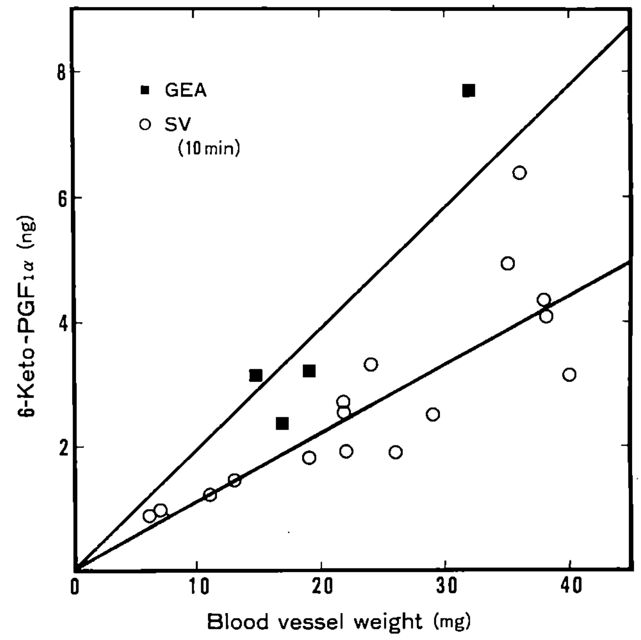

Fig 2. Production of 6-keto-prostaglandin $F_{1\alpha}$ (6-Keto-$PGF_{1\alpha}$) by gastroepiploic artery (GEA) and saphenous vein (SV) was positively linearly correlated with the vessel wet weight (GEA, n = 4; SV, n = 16, ten minutes of incubation).

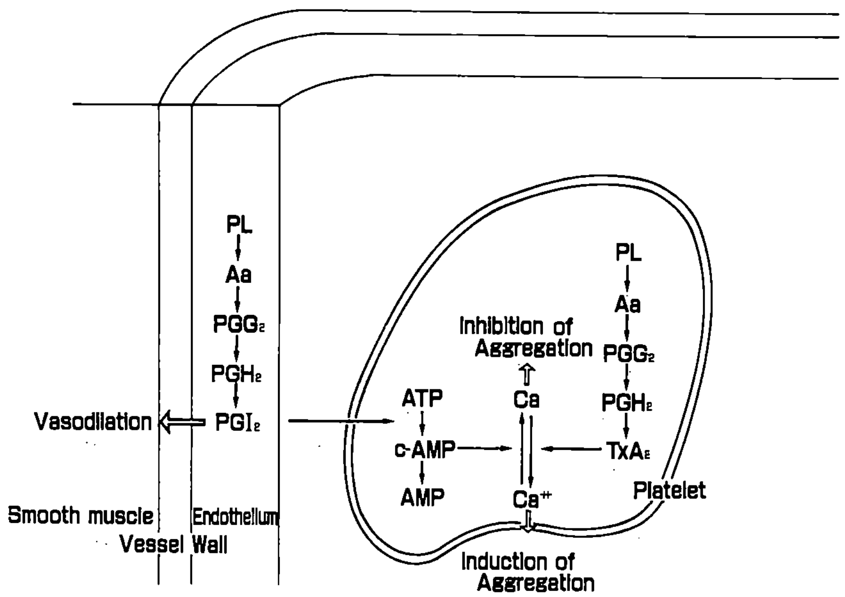

Fig 3. Prostacyclin (PGI$_2$) *is the major prostaglandin* (PG) *synthesized by endothelial cells. The precursor of prostacyclin is arachidonic acid* (Aa), *which is converted from phospholipids* (PL). *Prostacyclin is a potent inhibitor of platelet aggregation, and a vasodilator.* (AMP *= adenosine monophosphate;* cAMP *= cyclic adenosine monophosphate;* TxA$_2$ *= thromboxane A$_2$.*)

Chaikhouni and associates [8] reported that the basal production of 6-keto-PGF$_{1\alpha}$ by IMA was 152 ± 39 pg/mg tissue (mean ± standard error of the mean), whereas the SV produced only 68 ± 17 pg/mg tissue after 15 minutes of incubation in Krebs-Henseleit buffer at 37°C. Our results show that the basal production of 6-keto-PGF$_{1\alpha}$ by the GEA was 191.1 ± 21.8 pg/mg, whereas the SV produced 123.6 ± 15.1 pg/mg tissue after ten minutes. Chaikhouni and associates [8] attributed the proven higher patency rate of the IMA grafts over the SV to this higher production rate of prostacyclin by the IMA. The present comparative study of the GEA and the SV showed the same tendency, and has provided us a good reason to expect that GEA grafts will perform better than SV grafts and similar to IMA grafts.

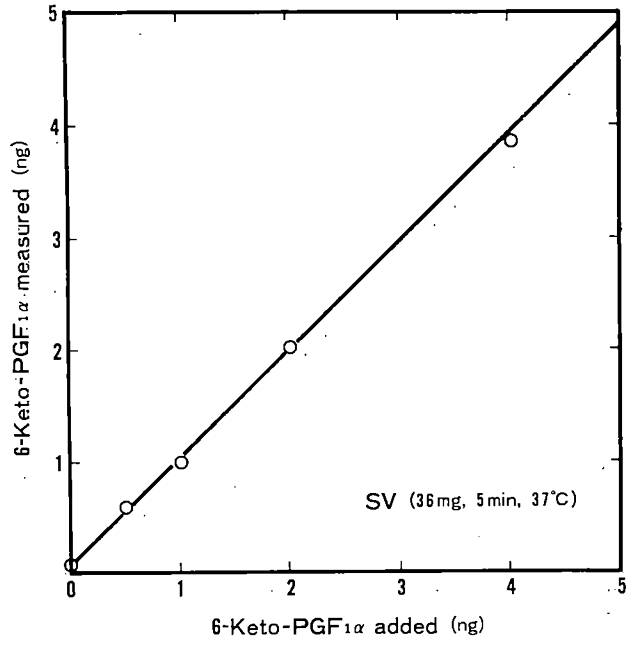

Fig 4. The measured 6-keto-prostaglandin F$_{1\alpha}$ (6-Keto-PGF$_{1\alpha}$) *is well correlated with the added 6-keto-PGF$_{1\alpha}$. The sample was 36 mg of saphenous vein* (SV) *at 5 minutes of incubation.*

Although the IMA graft has been recognized for its better patency than the SV graft, its harvest requires greater technical skill to avoid damage. The harvest of the GEA is easier than that of the IMA. The GEA is surrounded by loose, soft fat tissue and is easily isolated. The location and the course of the GEA can be traced easily by sensing its pulsation with one's fingers; thus the GEA can be more free from mechanical damage and is of better quality for bypass grafts as compared with the IMA.

Mills and Everson [18] described some criticisms such as: (1) the abdominal cavity must be entered, (2) the graft is not available after a gastric operation, (3) there is the possibility of graft damage with future abdominal procedures, (4) the long-term gastric or nutritional ill effects are unknown, and (5) unrecognized abdominal aortic or celiac atherosclerosis is difficult to assess. We must retain these factors in mind. However, our results suggest that the GEA will show a higher patency rate than the SV graft, and this is the greatest advantage of the bypass graft. The GEA appears to be a promising bypass graft material and an excellent graft for coronary artery revascularization.

We thank Dr Hiroaki Harasaki for thoughtful review of the manuscript and Noriko Ishikawa for secretarial assistance.

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Selective Approach to Sternal Closure After Exploration for Hemorrhage Following Coronary Artery Bypass

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Thirty-six (4.6%) patients required exploration for hemorrhage after 788 coronary artery bypass grafting procedures. Twenty-three (64%) patients with a specific site of bleeding that was surgically controlled or with improving coagulopathy were managed by immediate sternal closure. Continued hemorrhage or tamponade necessitated reexploration in 5 of these patients. All 5 patients were then treated by open sternotomy and delayed sternal closure. There were no deaths or sternal wound infections in this subgroup. Thirteen (36%) patients explored for hemorrhage were initially treated by open sternotomy and delayed sternal closure because of ongoing coagulopathy with refractory bleeding. Twelve patients recovered without further complication. One patient died 30 days after delayed sternal closure. There were no sternal wound infections. This experience supports a selective approach to sternal closure after exploration for hemorrhage following coronary artery bypass grafting. Immediate closure is recommended if a specific site of bleeding can be located and corrected. However, in the presence of refractory hemorrhage due to coagulopathy, delayed sternal closure should be considered to avoid the subsequent morbidity of continued bleeding, including cardiac tamponade, multiple reexplorations with sternal trauma, and retained mediastinal hematoma.

(Ann Thorac Surg 1990;49:771–4)

Postoperative hemorrhage continues to be a challenging complication of cardiovascular procedures. Bleeding after coronary artery bypass grafting (CABG) may be the result of coagulation derangement or technical causes. Early exploration is mandated by evidence of cardiac tamponade, hypovolemic shock, or unrelenting hemorrhage through drainage catheters. Management of the sternal wound after exploration for hemorrhage may also require surgical judgment. We have applied a selective approach to sternal closure after hemorrhage following CABG. An analysis of our experience with both primary and delayed sternal closure was undertaken to better define the indications and results of these techniques.

Material and Methods

During the 3-year period ending December 1987, 788 consecutive patients underwent CABG at Gundersen/Lutheran Medical Center, La Crosse, WI. Preoperative parenteral vancomycin was administered to all patients. At the termination of each procedure an Axion Mediastinal Silicone Drain (Axion, Paramount, CA) was placed substernally. The performance of single or bilateral internal mammary artery grafting required drainage of the ipsilateral pleural space. The sternum was reapproximated with No. 5 stainless steel wire, subcutaneous tissue with 2-0 polydioxanone suture, and skin with a running, subcuticular 4-0 undyed polyglycolic acid suture.

A standard postoperative coagulation profile including prothrombin time, partial thromboplastin time, and platelet count was sequentially determined, and attempts to correct specific abnormalities were initiated. Intravenous desmopressin acetate (0.3 μg/kg) was administered to all patients with postoperative bleeding. This dose was repeated six hours later if indicated. Positive end-expiratory pressure was occasionally used according to surgeon preference.

Exploratory sternotomy was performed for postoperative bleeding when the rate of blood loss exceeded 200 mL per hour or total blood loss approached 1,500 mL without evidence of slowing. Patients with hemodynamic instability, marginal cardiac output with continued bleeding, or evidence of cardiac tamponade were returned to the operating room more promptly. Exploratory sternotomy was performed in the operating room whenever possible. Clots were evacuated from the mediastinum. Cardiac suture lines were carefully inspected as well as each graft. If a specific bleeding site was identified and corrected or coagulopathy was improving, the sternum was immediately reclosed as previously outlined. In the presence of ongoing coagulopathy or continued hemorrhage from an indeterminate source, the sternotomy incision was left open with drains replaced into the mediastinum and pleural cavity as indicated. Sterile gauze laparotomy

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Table 1. Incidence of Exploratory Sternotomy for Hemorrhage After Coronary Artery Bypass Grafting in 788 Patients

Myocardial Revascularization	No. of Patients	Explored for Bleeding (%)
Vein graft	156	4 (2.6)
Internal mammary artery graft		
Unilateral	293	17 (5.8)
Bilateral	254	13 (5.1)
Repeat sternotomy		
Unilateral IMA	28	1 (3.6)
Bilateral IMA	26	1 (3.8)
Vein	31	0 (0.0)
Total	788	36 (4.6)

IMA = internal mammary artery.

packs were placed over the open wound and covered by an occlusive foam-tape dressing. The mediastinal tube was then placed on high-grade wall suction to prevent occlusion by blood clots. After correction of coagulation abnormalities and cessation of hemorrhage, delayed sternal closure was performed in the operating room. The sternum was reclosed in standard fashion using No. 5 stainless steel wire. Prophylactic parenteral vancomycin administration was begun before the original operation and continued until the sternum was closed and all mediastinal drains were removed.

Hospital charts were reviewed for total blood loss, coagulation variables (prothrombin time, partial thromboplastin time, platelet count), and time from operation to exploration. Discovery of a specific bleeding site on exploration was recorded, as well as the time from the exploration to delayed sternal closure. Complications noted in these patients included sternal wound infection, sternal dehiscence, and death.

Statistical comparisons of results between groups and means were made using χ^2 analysis of two proportions with Yates' correction and Student's t test, respectively. The level of confidence was defined at $p < 0.05$.

Results

Between January 1985 and December 1987, 788 patients underwent CABG at Gundersen/Lutheran Medical Center. Coronary artery bypass using vein grafts alone was performed in 187 (24%) patients and internal mammary artery grafts were used in 601 patients. Eighty-five patients (10.8%) had undergone previous sternotomy for a cardiac procedure. Cardiopulmonary bypass was performed in all cases using a membrane oxygenator.

During the 3-year study period, 36 patients (4.6%) required exploratory sternotomy for postoperative bleeding (Table 1). The study group included 24 men (67%) and 12 women (33%); ages ranged from 31 to 79 years (mean, 60 years). Cardiopulmonary pump times during the original operation ranged from 60 to 171 minutes (mean, 82 minutes). Exploration for bleeding was required in 18 (5.6%) of 321 patients undergoing CABG with a single internal mammary artery graft, and 14 (5.0%) of 280 patients with bilateral internal mammary artery grafts. Four explorations (2.1%) were performed in 187 patients after CABG with vein grafts alone. Two patients (2.4%) required exploration for bleeding after repeat sternotomy. No significant differences in the incidence of hemorrhage between these groups was determined.

Immediate Sternal Closure

Twenty-three (64%) patients were managed by immediate reclosure of the sternum after exploration. Average blood loss before exploration was 1,629 mL (range, 370 to 3,435 mL). Time from CABG to exploration ranged from one to 24 hours (mean, 7.5 hours). In 17 of these 23 patients the precise site of bleeding was found and corrected. No further complications developed in 16 of these patients. One patient continued to bleed secondary to worsening coagulopathy requiring a second exploration. This patient was then managed by open sternotomy and delayed sternal closure. In 6 of 23 patients a specific site of bleeding was not found. However, in the attending surgeon's judgment, coagulopathy was improving, hemorrhage was abating, and the sternotomy was closed primarily. Four of these 6 patients required a second exploration for continued hemorrhage or tamponade. All 4 were then managed by open sternotomy and delayed sternal closure.

Of the 18 patients treated with immediate sternal closure after exploration for hemorrhage who experienced no further bleeding, sternal wound infection developed in 2 and 1 died. The 17 survivors were discharged 7 to 37 days (median, 13 days) after exploration. All 5 patients who required a second exploration for bleeding after immediate sternal closure were then managed by open sternotomy and delayed sternal closure. Definitive closure was performed one to two days (mean, 1.8 days) later. Sternal wound infection did not develop in any of these patients. Nonseptic sternal bone dehiscence developed in 1 patient. There were no deaths.

Delayed Sternal Closure

Thirteen (36%) patients were managed by delayed sternal closure after exploration for hemorrhage. Average blood loss before exploration was 1,892 mL (range, 975 to 3,250 mL). Time from CABG to exploration ranged from 2.5 to 12 hours (mean, six hours). Diffuse, refractory bleeding from ongoing coagulopathy was evident in all 13 patients. Although a specific bleeding point was located and corrected in 2 individuals, concurrent diffuse hemorrhage with coagulopathy led the attending surgeon to elect open sternotomy and delayed sternal closure. Delayed closure was performed one to two days (mean, 1.3 days) after exploration. In no patient did sternal wound infection or dehiscence develop. One patient died within 30 days of exploration. The length of stay for the 12 survivors ranged from 8 to 21 days (median, 13 days) after exploration.

Comment

Serious postoperative hemorrhage has been reported in 0.5% to 25% of patients after CABG [1–4]. Poor hemostasis, imprecise suture lines, or other technical failures may be responsible. Hemorrhage may also be caused by coagulopathy associated with cardiopulmonary bypass. Frequently reported explanations for coagulopathy include clotting-factor consumption, heparin rebound, inadequate heparin reversal, thrombocytopenia, platelet dysfunction, and hypothermia [1–3, 5, 6]. Initial efforts at treating hemorrhage after CABG should focus on correction of these coagulation abnormalities by appropriate blood component and factor administration, correction of hypothermia, and normalization of acid-base balance. We have also administered desmopressin acetate to stimulate the endogenous release of Von Willebrand factor to enhance platelet function [7–9]. After correction of specific coagulation deficits, continued hemorrhage may require more direct techniques for control. Persistent hemorrhage from small chest wall and mediastinal vessels has been successfully controlled by increasing mediastinal pressure with positive end-expiratory pressure [4].

Failure of nonoperative techniques to adequately control hemorrhage after CABG mandates exploratory sternotomy. Bleeding of more than 400 mL in the first two to three hours after CABG in the presence of a normal coagulation profile warrants exploration [3, 5]. Others have used a rate of 100 to 200 mL per hour or 1,500 mL total postoperative blood loss as the threshold for exploration [2]. We have adopted the latter guidelines but support prompt exploration in the presence of hypovolemic shock, marginal cardiac output, or signs of cardiac tamponade. Timely intervention with rapid correction of bleeding from suture line disruption, cannulation sites, vein or internal mammary artery grafts, or the chest wall may prevent irreversible shock, cardiac decompensation, or complications associated with massive blood transfusions.

Optimal management of the sternal wound after exploration for hemorrhage is essential. Increased rates of sternal wound infection and bone dehiscence have been reported after exploratory sternotomy for bleeding following CABG [10, 11]. Other considerations related to wound closure include the risk of recurrent hemorrhage and cardiac tamponade. We have adopted a selective approach to sternal closure after exploration for bleeding following CABG (Fig 1). If a specific site of bleeding was located and definitively controlled, no further bleeding after immediate sternal closure was evident in 16 (94%) of 17 patients. However, in the face of worsening coagulopathy or hemorrhage from nonspecific sources, open sternotomy and delayed sternal closure has proved to be a valuable alternative. The principle of delayed sternal closure after cardiac operations has previously been applied for a variety of indications, including enlarged heart size with symptoms of tamponade on mediastinal closure, poor lung compliance, hemodynamic instability due to intractable arrhythmias, and need for a ventricular assist device [12–15]. This method of wound management has not been associated with increased rates of sternal wound infection [12, 14, 15]. We found no difference in the rate of sternal wound infection or dehiscence when comparing immediate and delayed closure after exploration for bleeding following CABG.

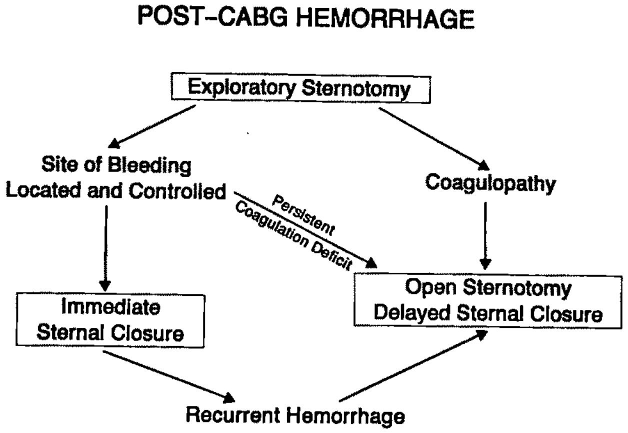
POST-CABG HEMORRHAGE
Exploratory Sternotomy
Site of Bleeding Located and Controlled
Coagulopathy
Persistent Coagulation Deficit
Immediate Sternal Closure
Open Sternotomy Delayed Sternal Closure
Recurrent Hemorrhage

Fig 1. Selective management of sternal wound closure after exploration for hemorrhage following coronary artery bypass grafting (CABG).

This experience supports a selective approach to sternal closure after exploration for hemorrhage following CABG. Immediate sternal closure was successful if a specific site of bleeding was located and corrected. In the presence of ongoing coagulopathy, open sternotomy and delayed sternal closure was effective in preventing the subsequent morbidity of continued hemorrhage, including cardiac tamponade, multiple reexplorations with repeated sternal trauma, and retained mediastinal hematoma.

We gratefully acknowledge the financial support of the Gundersen Medical Foundation.

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REVIEW OF RECENT BOOKS

Pediatric Brain Death and Organ/Tissue Retrieval. Medical, Ethical, and Legal Aspects

Edited by Howard H. Kaufman
New York, Plenum, 1989
365 pp, illustrated, $49.50

Reviewed by Michael P. Kaye, MD

This comprehensive work on organ donations is based on a conference held in March 1987. Participants in the conference covered in detail all aspects of organ donation in children including the historical, ethical, and legal considerations. Detailed analysis of criteria for brain death were described and documented, and current and future needs in diverse areas were discussed in detail.

The book is of clear benefit in establishing a background of factual information in this most controversial area and would prove to be a worthwhile addition to the library of those who in any way are associated with organ donations. All religious, ethical, and legal facets are covered in detail. There is an additional section devoted to societal issues including the short-term and long-term effects on family. Medical criteria of brain death are presented in detail. Finally, a chapter is devoted to the federal efforts involved in the enhancement of the organ procured system.

Although this book would be a worthwhile addition to the library of anyone associated with the field of transplantation from a historical and reference standpoint, changes in this area of transplantation are occurring so rapidly that the "latest" information and attitudes presented in 1987 are now well outdated. Finally, a slightly larger print size would make this volume much easier to read.

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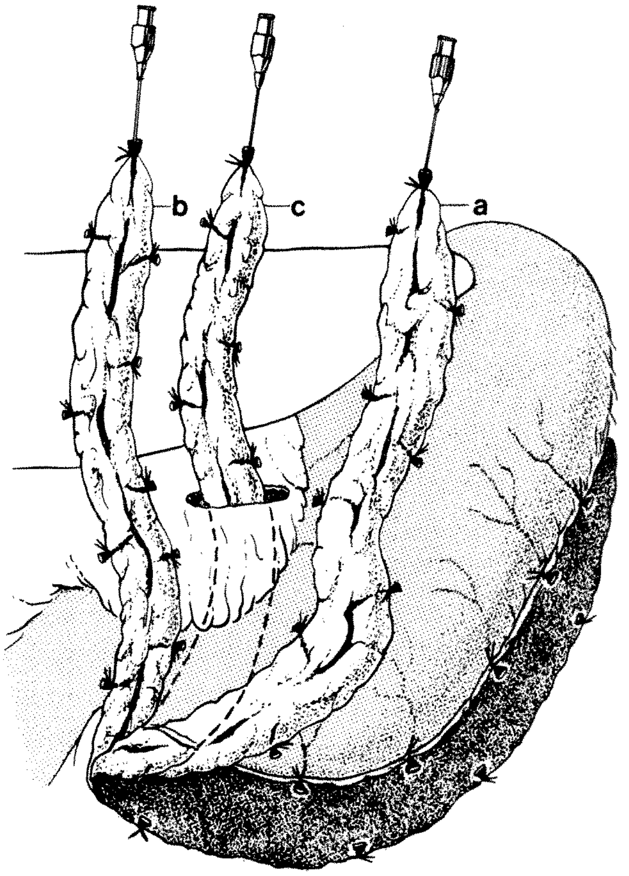

Figure 4. *Routes of the right gastroepiploic graft to the diaphragm are:* **(A)** *anterior to the stomach (rare),* **(B)** *anterior to the duodenum, and* **(C)** *posterior to the stomach through the avascular space in the lesser sac (most common).*

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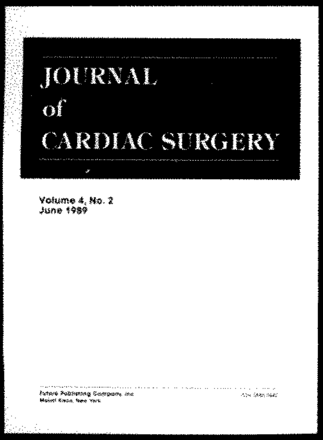

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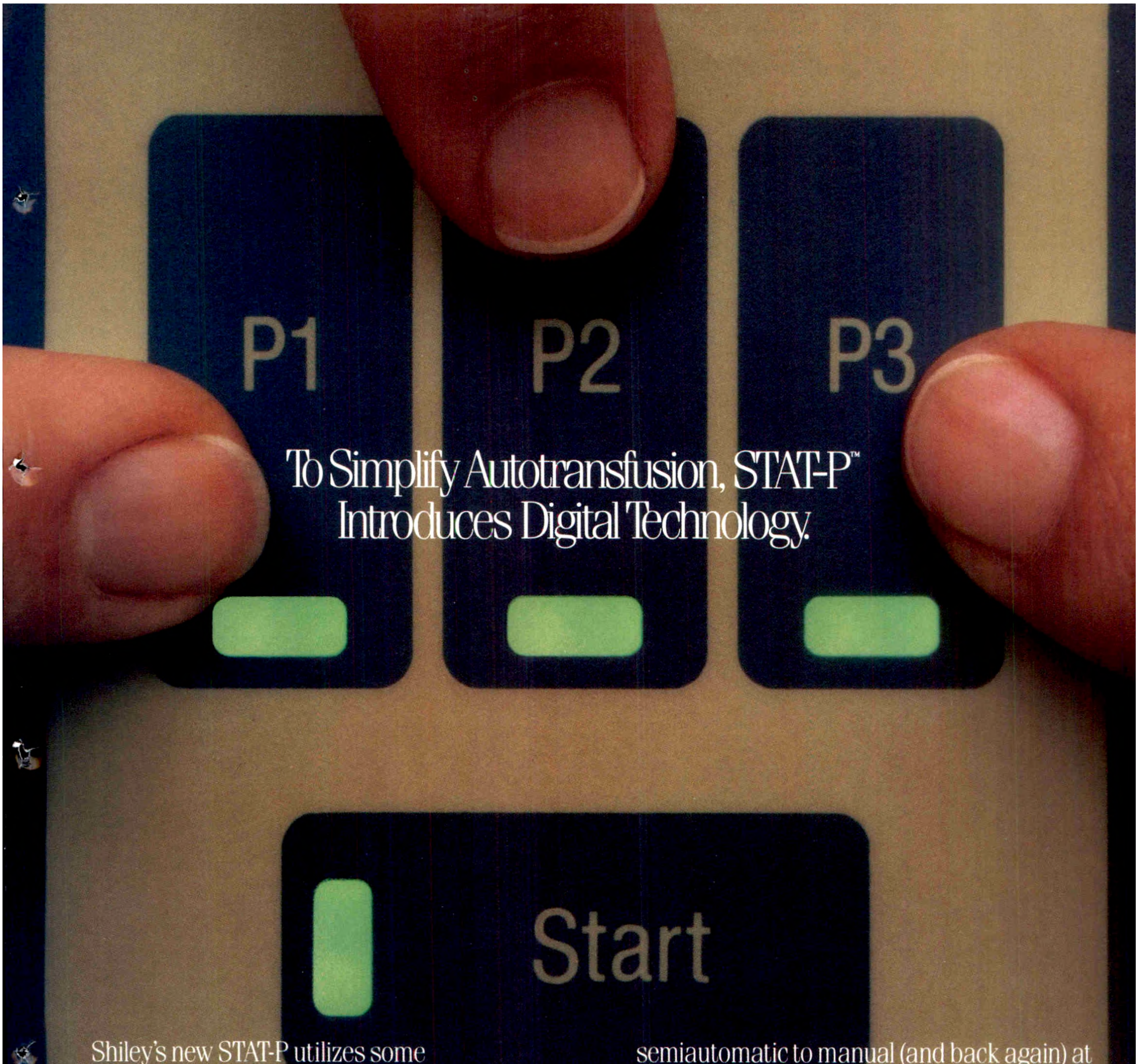

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1. Heinemann F, et al: Clinical comparison of available 'low threshold' leads. European Society of Cardiology, Congress, Vienna, 1988.
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3. IS-1 is the designation of the standard pacing connector developed by the International Committee on Cardiovascular Implants for Surgery (International Standard Organization ISO TC150/SC).

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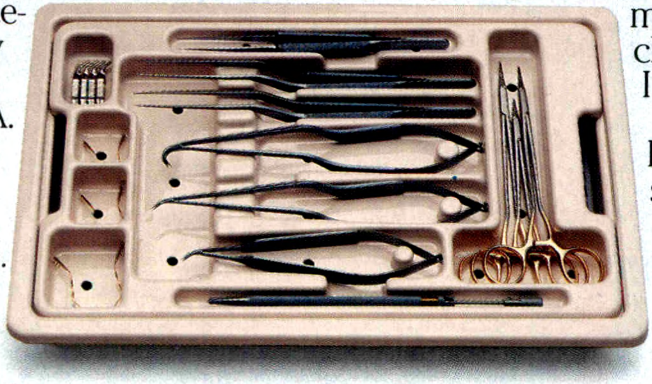

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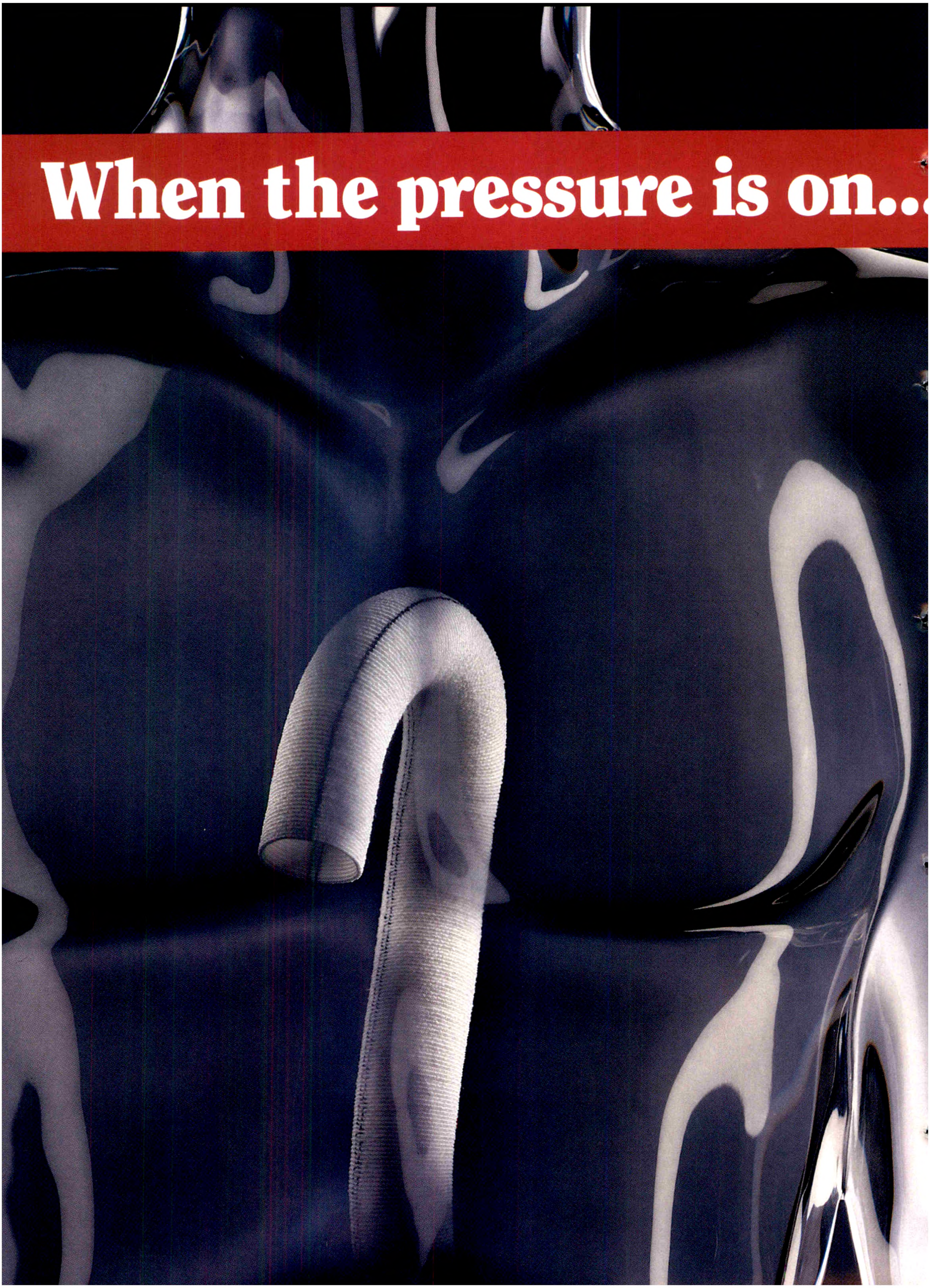
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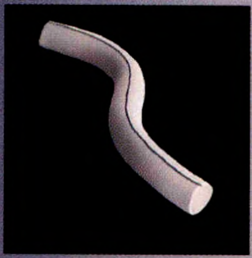

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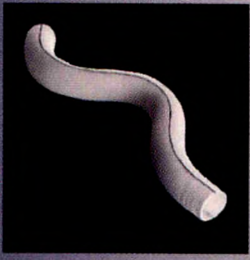

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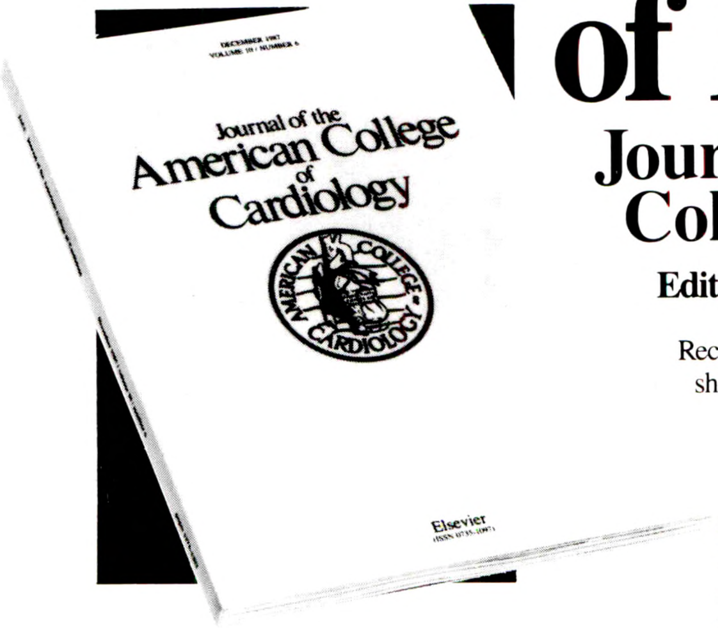

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Palliative Reconstruction of Right Ventricular Outflow Tract in Tetralogy With Hypoplastic Pulmonary Arteries

Yutaka Okita, MD, Shigehito Miki, MD, Kenji Kusuhara, MD, Yuichi Ueda, MD, Takafumi Tahata, MD, Kazuo Yamanaka, MD, and Tokio Tamura, MD

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Twenty-five symptomatic patients with tetralogy of Fallot underwent palliative reconstruction of the right ventricular outflow tract without closure of the ventricular septal defect. Their ages ranged from 5 months to 20 years (mean age, 3.5 years). Eight patients had had 13 prior systemic–pulmonary arterial shunts. There were six hospital deaths (24%). Three patients needed a repeat right ventricular outflow reconstruction. There was one late death. The other patients manifested clinical improvement; hematocrit decreased from 0.54 to 0.43 ($p < 0.01$) and arterial oxygen saturation increased from 63.0% to 83.2% ($p < 0.01$). The size of the pulmonary artery, defined as the ratio of the sum of the diameter of the right and left pulmonary arteries to the diameter of the descending aorta, increased from 0.72 to 2.06 ($p < 0.01$). Sixteen patients underwent a corrective operation 2.4 years after palliation. The results of palliative right ventricular outflow tract reconstruction suggest that it may be an optional strategy for the treatment of symptomatic patients who have tetralogy of Fallot with severely hypoplastic pulmonary arteries.

(Ann Thorac Surg 1990;49:775–9)

The surgical treatment of symptomatic tetralogy of Fallot requires a choice between total correction or palliation. Some patients with tetralogy appear to be poor candidates for initial total correction because of poor development of the pulmonary arterial tree. Controversy exists concerning the identification of these patients and the best form of palliation to use. At Tenri Hospital in Nara, Japan, the treatment for select patients with symptomatic tetralogy with hypoplastic pulmonary arteries has been to undertake palliation by enlarging the right ventricular outflow tract and main pulmonary artery, while leaving the ventricular septal defect unclosed.

Patients and Methods

From June 1981 through July 1988, 25 symptomatic patients with tetralogy of Fallot underwent palliative reconstruction of right ventricular outflow tract without closure of the ventricular septal defect. The patients were chosen on a highly selective basis, because of severely hypoplastic pulmonary arteries, failure of previously constructed systemic–pulmonary arterial shunts, or branch pulmonary arterial stenosis. There were 13 male and 12 female patients, with ages ranging from 5 months to 20 years (mean age, 3.5 years). All patients had signs and symptoms of inadequate pulmonary blood flow, including persistent cyanosis and decreased exercise tolerance. Thirteen systemic–pulmonary arterial shunts (a Blalock-Taussig shunt in 8 patients and a central shunt in 5) had previously been performed on 8 patients. The mean preoperative hematocrit value was 0.54 ± 0.09, and the mean arterial oxygen saturation ratio was 63.0% ± 10.1%. All patients underwent preoperative cardiac catheterization. The main pulmonary artery was atretic in 7 patients. In the patients with previous Blalock-Taussig anastomosis, the left pulmonary artery was occluded proximally in 2 and narrowed in 1 patient. Among the patients without a previous surgical shunt, atresia of the left pulmonary artery was noted in 1, and proximal stenosis of the left pulmonary artery was noted in 5. No patient had major aortopulmonary collateral arteries.

Measurements of the pulmonary arterial diameter were obtained from angiocardiogram with use of the anteroposterior view. The right and left pulmonary arteries were measured just proximal to the first segmental branches. The descending thoracic aorta was measured just above the diaphragm. For normalization of pulmonary arterial size to the size of the patient, measurements are expressed as a ratio of the summed diameter of the right and left pulmonary arteries to the diameter of the descending thoracic aorta (McGoon's ratio) [1]. Associated anomalies were persistent left superior vena cava in 5 patients, anomalous origin of left anterior descending coronary arteries in 1 patient, and common atrioventricular canal in 1. One patient suffered severe congestive failure due to depressed biventricular contractility. Evidence of mental retardation was detected in 4 patients.

All data are presented as the mean ± standard deviation. Serial data in each group were compared by use of paired Student's t test.

Accepted for publication Dec 29, 1989.

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Surgical Technique

All procedures were performed via median sternotomy using mild hypothermic cardiopulmonary bypass. A single venous cannula was introduced into the right atrium. During the earlier experience, two balloon occlusive catheters (Fogarty, Baxter Healthcare, Irvine, CA) were introduced into the proximal right ventricular cavity and into the distal pulmonary artery for the purpose of obtaining an asanguineous surgical exposure. Right ventricular outflow reconstruction was performed with the heart beating. More recently, cardiac arrest was obtained by induced fibrillation or aortic root injection of cold cardioplegic solution. A short longitudinal right ventricular incision limited to the infundibulum was made. In 18 patients, the ventriculotomy was extended across the pulmonary annulus onto the main pulmonary arterial trunk. The arteriotomy was extended out on the left pulmonary artery in 1 patient. In 1 patient, a 8-mm expanded polytetrafluoroethylene (Gore-Tex, Gore & Associates, Inc, Elkton, MD) conduit was used because of coronary arterial anomaly. A small amount of infundibular muscle was resected to create a channel 5 to 10 mm in diameter. In 10 patients, an equine pericardial patch was sutured to the epicardial edge of the ventriculotomy. A Dacron patch was used in 7 patients, an expanded polytetrafluoroethylene patch (Gore-Tex) in 7, and a porcine monocusp patch (MVOP, Polystan Co, Copenhagen, Denmark) in 1 patient. No attempt was made to reconstruct the completely occluded left pulmonary artery.

Results

There were six hospital deaths (24%). The cause of death was congestive heart failure due to excessive pulmonary blood flow in 2 patients (their McGoon's ratios were 0.9 and 1.5), a preexisting left ventricular dysfunction of unknown cause in 1, cerebral accidents in 2, and inadvertent injury to the left anterior descending coronary artery in 1 patient. However, among the last 10 patients, only one death (10%) has occurred. Three patients needed repeat right ventricular outflow reconstruction because of inadequate growth of the pulmonary arteries. There was one late death due to hypoxemia. This patient had undergone a right ventricular–pulmonary arterial conduit reconstruction because of an anomalous left anterior descending coronary artery, which was crossing the right ventricular outflow tract. Other patients manifested major clinical improvement; hematocrit decreased from 0.54 ± 0.09 to 0.43 ± 0.06 ($p < 0.01$) and arterial oxygen saturation increased from 63.0% ± 10.1% to 83.2% ± 10.5% ($p < 0.01$). Cardiac catheterization was performed 12 to 36 months (mean, 23.7 months) after the outflow reconstruction in 16 patients. The study revealed that the size of the pulmonary arteries (McGoon's ratio) increased from 0.72 ± 0.41 to 2.06 ± 0.47 ($p < 0.01$) (Figs 1–3). The ratio of the pulmonary blood flow to systemic flow ranged from 0.19 to 3.3 (mean, 1.41 ± 0.92), and the ratio of systolic pressure ratio of the pulmonary artery to the aorta was between 0.15 and 0.76 (mean, 0.35 ± 0.26).

Among 18 long-term survivors, 16 patients underwent total correction on an elective basis 2.4 ± 0.6 years after right ventricular outflow reconstruction. A transannular monocuspid patch was used in 10 patients, pulmonary valve replacement by xenograft (Carpentier-Edwards bioprosthesis, Baxter Healthcare, Irvine, CA) in 5, and infundibular patch in 1. The indication for prosthetic pulmonary valve insertion was the coexistence of severe pulmonary regurgitation and a peripheral pulmonary arterial stenosis or pulmonary hypertension. A pericardial patch plasty of an orifice stenosis of the left pulmonary artery was performed in 5 patients. After total correction, a peak systolic pressure ratio of the right ventricle to the left ventricle was 0.48 ± 0.38 and that of the right pulmonary artery to the left ventricle was 0.35 ± 0.21. One patient had persistent pulmonary hypertension with a pulmonary arterial pressure of 55 mm Hg. There was one death after correction due to low cardiac output syndrome. The patient had had a sustained right ventricular hypertension. The remaining 2 patients are waiting for total correction. All the patients who survived corrective operation were in New York Heart Association functional class I or II.

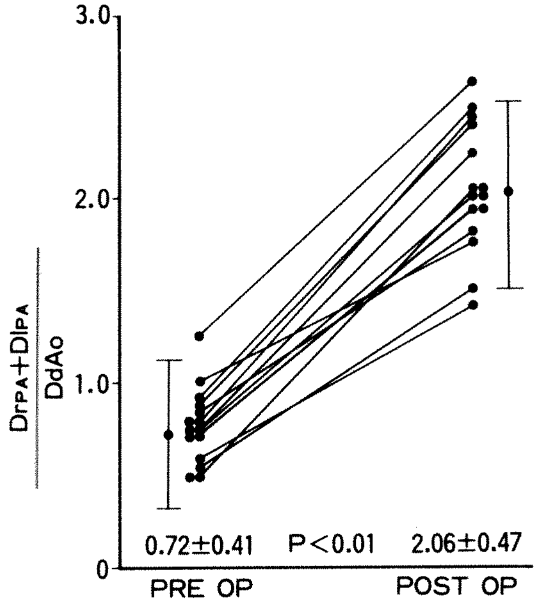

Fig 1. Mean and individual change in normalized pulmonary arterial diameter after right ventricular outflow tract reconstruction. (DrPA + DlPA / DdAo = *ratio of the sum of the diameter of the right and left pulmonary arteries to the diameter of the descending aorta.)*

Comment

Excellent clinical results of the correction of tetralogy of Fallot in infants were demonstrated by Barret-Boyes [2], Starr [3], and their associates in the early 1970s. More recently, Castaneda and associates [4] have substantiated this view of early correction with reports of outstanding results in a series in which total repair was performed for tetralogy in patients under the age of 1 year.

On the other hand, the subgroup of patients with tetralogy of Fallot who cannot tolerate total correction are infants with diminutive pulmonary arteries. Some inves-

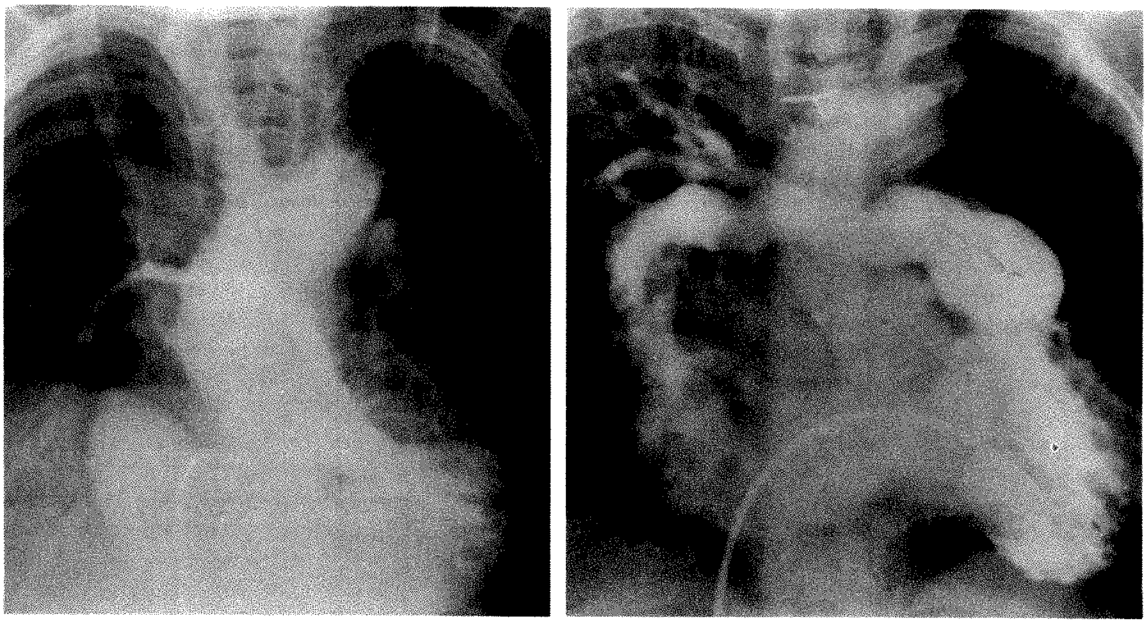

Fig 2. Right ventriculography in a patient with tetralogy before (left) and after (right) palliative right ventricular outflow tract reconstruction. The left pulmonary artery was occluded at the site of a previous Blalock-Taussig shunt. There is a marked increase in the size of the right pulmonary artery.

tigators [5, 6] have reported the criteria for selecting patients for total correction, emphasizing the importance of the size of the pulmonary arteries. Many surgical strategies to develop such diminutive pulmonary arteries have been investigated. Conflicting reports have appeared regarding the growth of pulmonary arteries after palliation with the systemic–pulmonary shunt. Gale and associates [7] have shown growth of the pulmonary annulus and pulmonary arteries after the Blalock-Taussig shunt was performed. Gill and co-workers [8], however, have expressed concern about the occurrence of kinking and stenosis when the Blalock shunt is performed on infants with diminutive pulmonary arteries. Lamberti and associates [9] reported a more symmetrical and greater increase in the diameter of the pulmonary artery after the central shunt than the Blalock-Taussig shunt. Regarding the ascending aorta–right pulmonary artery shunt (Waterson), a high incidence of kinking of the pulmonary artery is a well-documented phenomenon when this is performed in small infants [10, 11].

In 1948, Lord Brock reported 3 patients with tetralogy who successfully underwent closed transventricular pulmonary valvotomy [12]. Recently, Piehler [13], Freedom [14], Millikan [15], Kirklin [16], and their associates have reported excellent surgical results of right ventricular outflow tract reconstruction for symptomatic patients with tetralogy using cardiopulmonary bypass. The practical advantage of the palliative right ventricular outflow tract reconstruction is that it offers substantially greater, more rapid, more uniform, and more predictable pulmonary arterial enlargement and is associated with less pulmonary arterial distortion than the conventional shunt procedures. Piehler and associates [13] reported a 2.20-fold increase in mean pulmonary artery size 26 months after outflow tract reconstruction in 12 patients, which

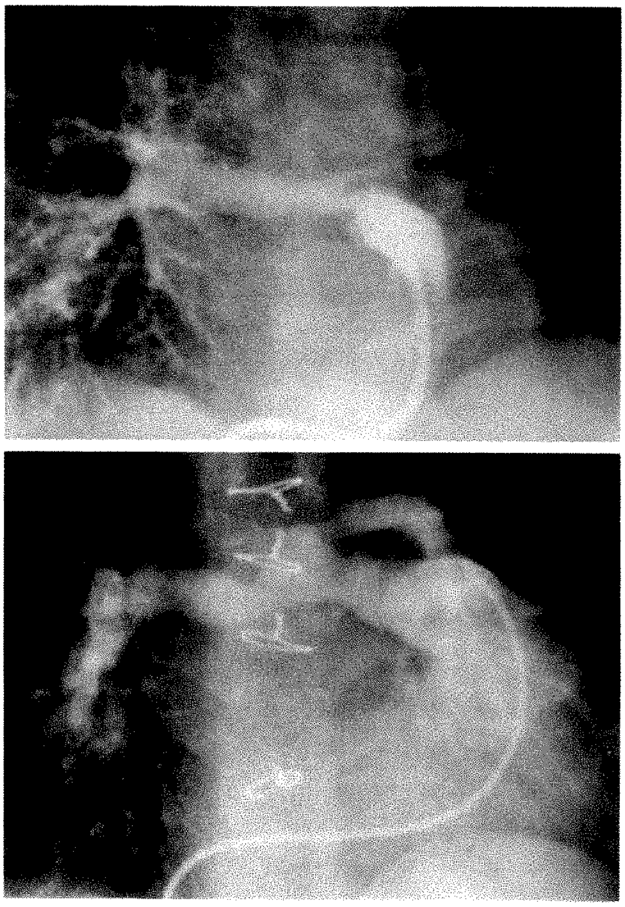

Fig 3. Right ventricular outflow tract injection in a patient with tetralogy before (top) and after (bottom) palliative right ventricular outflow tract reconstruction. Although the left pulmonary artery was severely hypoplastic, symmetrical growth of both pulmonary arteries was achieved.

was comparable with a 1.55-fold increase in the pulmonary artery size after shunt operation. Millikan and coworkers [15] reported that the average size of pulmonary arteries grew from 25% to 99% of normal after the palliative reconstruction of the right ventricle over a mean interval of 32 months. Similarly, Sebening and associates [17] reported that favorable growth of the pulmonary artery obtained by outflow reconstruction was superior to that from a closed pulmonary valvotomy or classic shunt operation. The increased pulmonary blood flow affords a more favorable and efficient arteriovenous admixture than provided by a systemic–pulmonary artery shunt.

A relative disadvantage of the outflow tract reconstruction procedure is the requirement for sternotomy and cardiopulmonary bypass. Although Freedom and colleagues [14] and Tucker and co-workers [18] reported excellent results of right ventricular outflow tract reconstruction in infants, the requirement for sternotomy and cardiopulmonary bypass might assume considerable importance in patients younger than 2 years of age. Piehler and associates [13] reported a high operative mortality in younger patients after right ventricular outflow tract reconstruction. Their cause of death was exclusively low cardiac output syndrome.

A second potentially lethal drawback is that outflow tract reconstruction may cause congestive heart failure due to excessive pulmonary blood flow. There were some difficulties in predicting the smallest pulmonary arterial size that was compatible with successful complete repair. In palliative reconstruction of the right ventricular outflow tract, pulmonary blood flow is determined primarily by pulmonary arterial size. This is in contrast to conventional shunts, in which blood flow is limited mainly by anastomotic and conduit size. Pulmonary arteries that are too large permit excessive pulmonary blood flow and could cause severe congestive heart failure after the outflow tract palliation. This phenomenon is remarkable especially among infants. Two infants died because of underestimation of the pulmonary artery size. However, with increasing experience with our surgical treatment of tetralogy in infancy and the application of the criteria [6, 19], determined by pulmonary arterial size, that predict correctability, we have reduced mortality and morbidity in this age group.

A third potential disadvantage of the outflow reconstruction is the development of pulmonary valve regurgitation. Some investigators [13, 18] are of the opinion that the large pulse wave seen in patients with pulmonary valvular regurgitation may be advantageous in enlarging the pulmonary arterial tree. However, most patients needed pulmonary valve replacement at the corrective operation because of severely damaged semilunar cusps, peripheral branch stenosis of the pulmonary arteries, and pulmonary hypertension. We believe that the additional risk of pulmonary valve replacement burdening the heart, although inevitable, is the price paid for relieving the pulmonary valvular regurgitation.

Fourth, Freedom and associates [14] expressed concern about the left pulmonary arterial stenosis caused by the outflow patch. Piehler and colleagues [13] warned that an excessively long arteriotomy and an unreasonably wide patch could generate distal anastomotic and left pulmonary arterial stenosis. In our series, 1 patient died after correction due to low cardiac output syndrome resulting from severe left pulmonary arterial stenosis. Among the survivors, the incidence of postoperative right ventricular hypertension is higher than that of the patients who underwent primary corrective repair. We consider the peripheral pulmonary arterial stenosis caused by the outflow patch to be one of the predisposing factors of the high right ventricular pressure after correction. Therefore, use of the outflow patch should be limited solely to relieving central stenosis of the right ventricular outflow tract.

Finally, this palliation can cause obstructive pulmonary arterial vascular disease when an excessive left-to-right shunt flow persists. With regard to the timing of the corrective operation after palliation, it depends on the developed size of the pulmonary artery and the predominant direction and magnitude of the shunt through the ventricular septal defect. A safer corrective repair should be assured when the blood flow through the right ventricular outflow tract becomes greater than 1.5 times systemic flow [13]. Generally, in younger patients, the greatest increase in pulmonary arterial growth occurs sooner after outflow reconstruction. Those infants should undergo catheterization within 6 months after this palliation and proceed to repair. We do not consider resternotomy to be a risk factor in correction of tetralogy.

Consequently, we made it our general guideline for symptomatic patients with tetralogy and markedly hypoplastic pulmonary arteries (McGoon's ratio less than 0.75), or with distorted pulmonary arterial trees, to establish systemic–pulmonary arterial shunts in infants, especially if less than 6 months of age, and perform palliative reconstruction of the right ventricular outflow tract in older children.

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Descending Necrotizing Mediastinitis: Transcervical Drainage Is Not Enough

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One of the most lethal forms of mediastinitis is descending necrotizing mediastinitis, in which infection arising from the oropharynx spreads to the mediastinum. Two recently treated patients are reported, and the English-language literature on this disease is reviewed from 1960 to the present. Despite the development of computed tomographic scanning to aid in the early diagnosis of mediastinitis, the mortality for descending necrotizing mediastinitis has not changed over the past 30 years, in large part because of continued dependence on transcervical mediastinal drainage. Although transcervical drainage is usually effective in the treatment of acute mediastinitis due to a cervical esophageal perforation, this approach in the patient with descending necrotizing mediastinitis fails to provide adequate drainage and predisposes to sepsis and a poor outcome. In addition to cervical drainage, aggressive, early mediastinal exploration—debridement and drainage through a subxiphoid incision or thoracotomy—is advocated to salvage the patient with descending necrotizing mediastinitis.

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Mediastinitis occasionally develops as a complication of cervical or odontogenic infections. Best referred to as descending necrotizing mediastinitis (DNM), such infections usually have a fulminant course, leading to sepsis and frequently death. The diagnosis of DNM is almost always delayed and is usually made when sepsis develops after seemingly adequate surgical drainage of the cervical infection. For most patients at this point, drainage of the superior mediastinum through a cervical incision is inadequate. In a recent comprehensive review of this topic, Estrera and associates [1] documented a 40% mortality rate in the postantibiotic era for patients with this type of lethal mediastinitis. Despite these authors' recommendations for early diagnostic computed tomographic (CT) scanning for all cervical infections, as well as for aggressive mediastinal drainage including thoracotomy if necessary, published reports since 1983 continue to document very high mortality rates for DNM. Two recent patients treated for DNM at our institution have emphasized the absolute importance of diagnostic CT scanning and aggressive mediastinal drainage in the survival of these patients.

Case Reports

Patient 1

A 39-year-old man complained of left mandibular pain and was started on oral penicillin. Within 24 hours, a fever to 39°C and marked facial swelling developed, prompting admission to the University of Michigan Medical Center. Physical examination revealed induration of the anterior neck, trismus, and tongue elevation. He underwent third molar extraction, tracheostomy, and bilateral submandibular and submental drainage. Cultures revealed a mixed aerobic and anaerobic infection. Despite drainage and intravenous penicillin and metronidazole, he remained febrile and had purulent drainage from his wounds. On hospital day 4, substernal chest pain developed, accompanied by nonspecific electrocardiographic changes and a left pleural effusion on chest roentgenogram. Respiratory insufficiency ensued shortly thereafter and, on hospital day 6, a CT scan demonstrated gas collections in the anterior and posterior mediastinum (Fig 1). Needle thoracentesis of the left pleural fluid revealed 1.5×10^5 white blood cells per liter but culture was sterile. After thoracic surgical consultation, the neck was reexplored with the finding of pus in all fascial planes, which was widely drained. Anterior mediastinal drainage was accomplished through a subxiphoid counterincision through which a large sump drain was placed posterior to the sternum to the level of the neck. The upper posterior mediastinum was drained through the cervical incision. After initial improvement, fever again developed on hospital day 10, prompting reexploration of the mediastinum through the cervical and subxiphoid incisions. The anterior mediastinum was again approached through the subxiphoid incision and blunt manual dissection was used to more widely open the tissue planes for better drainage. After this procedure, the patient's condition steadily improved. His anterior mediastinal drains were removed on hospital day 19 and, after completion of antibiotic treatment, he was discharged on hospital day 36. He continued to do well 7 months postoperatively.

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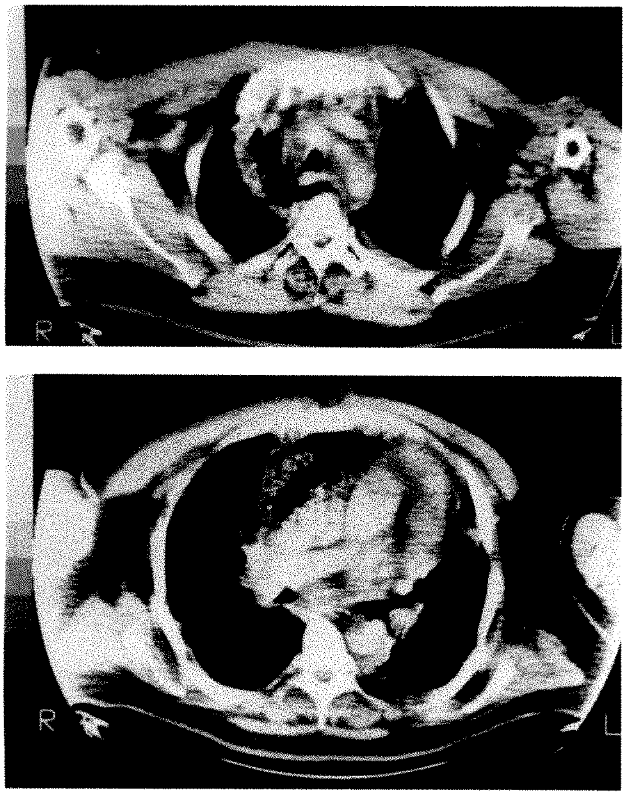

Fig 1. Chest computed tomographic scan showing a gas-forming infection involving the upper mediastinum and the anterior mediastinum well below the carina.

Patient 2

A 26-year-old man underwent extraction of his upper and lower right third molars. Two days later, soreness developed in the right anterior neck that was unresponsive to treatment with oral penicillin. The patient was seen with dysphagia and pleuritic chest pain. Physical examination revealed trismus and diffusely tender cervical swelling. A chest roentgenogram revealed possible air anterior to the trachea, and CT scan confirmed the presence of paratracheal air and suggested mediastinitis. Intravenous penicillin and metronidazole were begun, and he then underwent tracheostomy and neck drainage through bilateral cervical incisions. The pretracheal, parapharyngeal, and carotid spaces were all infected, cultures revealing β-hemolytic streptococcus and several anaerobic organisms. He remained febrile and, on hospital day 2, a follow-up CT scan confirmed mediastinitis with fluid and air in the mediastinum. He was returned to the operating room where his neck was reexplored and superior mediastinal drains were placed. Then, through a right anterolateral thoracotomy, debridement and drainage of the right paratracheal space were performed. Postoperatively, he required two additional cervical drainage procedures for a recurrent right posterior triangle abscess, but his condition steadily improved. His chest tubes were slowly advanced and removed on hospital day 28. The following day, administration of antibiotics was stopped, and he was discharged. He is doing well 4 months postoperatively.

Table 1. Causes of Descending Necrotizing Mediastinitis

Cause	No. of Patients
Odontogenic infections	25
Retropharyngeal abscess	6
Peritonsillar abscess	2
Cervical lymphadenitis	2
Trauma	3
Endotracheal intubation	4
Unknown	1
Total	43

Review of the Literature

All published reports from 1960 to the present on descending necrotizing mediastinitis arising from an oropharyngeal source were reviewed with the aid of the *Index Medicus* and the Medline computer search system. Previously established criteria for the diagnosis of DNM were used, and include: (1) clinical manifestations of oropharyngeal infection (odontogenic, peritonsillar, or retropharyngeal abscesses, Ludwig's angina, or infection secondary to traumatic pharyngeal perforations); (2) characteristic roentgenographic features of mediastinitis; (3) documentation of mediastinitis at operation or postmortem examination; and (4) establishment of a relationship between oropharyngeal infection and subsequent necrotizing mediastinitis [1]. Cases of mediastinitis secondary to cervical esophageal perforations were not included in this review as the diagnosis, treatment, and clinical course of this type of suppurative mediastinitis have been well established [2–5].

Since 1960, a date well into the postantibiotic era, 43 cases of DNM have been reported [1, 6–29]. The group consists of 32 male and 9 female patients, with the sex of 2 patients not reported. The average patient age is 32 years, ranging from 1 month to 64 years. The causes of infection from which mediastinitis developed are shown in Table 1. The most common cause, accounting for over half of all cases of DNM, is odontogenic infection, usually arising from the second or third molar. Bacteriologic analysis revealed mixed aerobic and anaerobic organisms for most patients, although β-hemolytic streptococcus was the sole pathogen for 5 patients (Table 2). All patients were initially treated with antibiotics and cervical drainage for the oropharyngeal infection. The surgical procedures used for mediastinal drainage are listed in Table 3. The majority of patients underwent initial superior medi-

Table 2. Bacteriology of Descending Necrotizing Mediastinitis

Organism	No. of Patients
Aerobic (β-hemolytic streptococcus)	5
Anaerobic	1
Mixed aerobic and anaerobic	30
Not reported	7
Total	43

Table 3. Surgical Procedures for Descending Necrotizing Mediastinitis

Procedure	No. of Patients
Cervicomediastinal drainage	12
Cervical drainage and simultaneous thoracotomy	2
Cervicomediastinal drainage with subsequent thoracotomy necessary for mediastinal drainage	20
No treatment—postmortem diagnosis	6
Unknown	3
Total	43

astinal drainage through a cervical approach. Although 2 patients underwent simultaneous cervical drainage and thoracotomy, 19 patients treated initially with transcervical drainage required a subsequent thoracotomy because of inadequate control of the mediastinal infection. Overall, transcervical mediastinal drainage of DNM provided incomplete or inadequate drainage in 26 of 33 patients so treated. Further, several authors [1, 8, 15, 24] noted that transcervical mediastinal drainage often delayed more definitive operation until overwhelming sepsis ensued. The overall reported mortality for DNM since 1960 has been 36% (15/42). The morbidity for survivors was also high, with patients undergoing an average of three surgical procedures and having an average hospital stay of 37 days.

In considering the reported mortality for DNM, it is instructive to contrast the results of treatment of this condition before and after 1983. In spite of the publication of two comprehensive reports on DNM in 1983 documenting the utility of early CT scanning to diagnose mediastinitis and, most importantly, the absolute necessity of early and complete mediastinal drainage with thoracotomy when required [1, 24], the 10 published cases since 1983 [25–29] still record a 40% mortality, similar to the 33% rate before 1983. Further, in 7 of the 10 cases treated since 1983, initial cervical drainage of the mediastinum failed, similar to the 80% failure rate reported before 1983.

Comment

The most common underlying cause for mediastinitis in the postantibiotic era is esophageal disruption. A much less common but perhaps more virulent form of mediastinal infection is DNM, in which infections originating in the fascial spaces of the head and neck dissect downward into the mediastinum. Despite the increased use of the CT scan as a diagnostic aid and the improvement in antibiotic regimens, DNM remains lethal in at least one third of patients.

An understanding of the anatomy of the cervicomediastinal fascial spaces and planes is essential to the surgical management of this disease, and several detailed reviews of this subject are available [2, 3, 13, 30, 31]. Briefly, the deep cervical fascia is arranged into three layers: a superficial layer, a visceral layer, and a prevertebral layer. These layers partition the neck into three potential spaces, which can all serve as portals of entry into the mediastinum (Fig 2).

The potential space in front of the trachea and posterior to the strap muscles is the pretracheal space. Perforations of the trachea and lateral pharyngeal walls can give rise to infections in this space. The pretracheal fascia has attachments to the pericardium and the parietal pleura at the level of the carina, providing an explanation for the findings of purulent pericarditis and empyema often seen with DNM.

The retrovisceral space lies behind the esophagus and anterior to the prevertebral fascia and extends upward to the base of the skull and inferiorly into the posterior mediastinum. This is the most common route by which oropharyngeal infections spread to the mediastinum.

The perivascular compartment is surrounded by the carotid sheath, which represents the fusion of the major layers of the cervical fascia. Infections can follow the vascular structures into the mediastinum and pleural space.

Odontogenic and peritonsillar abscesses rupture into the submandibular and parapharyngeal spaces. The parapharyngeal space occupies a critical position in the neck, communicating with all major fascial spaces [6]. Parapharyngeal abscesses readily extend into the retropharyngeal space along the styloglossus muscle, providing the major

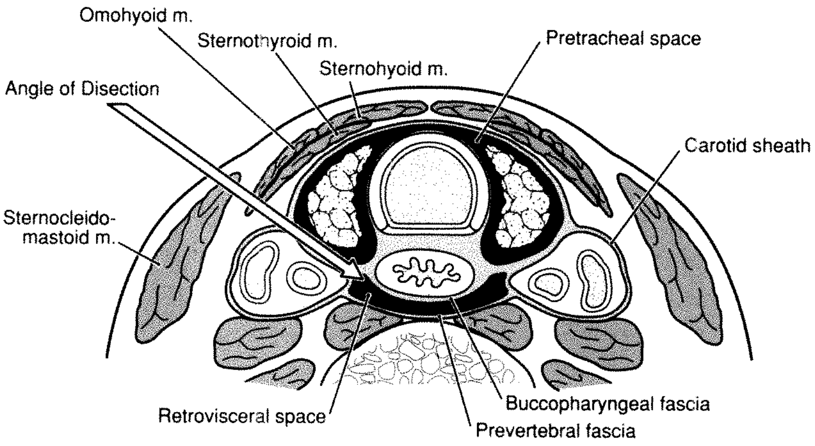

Fig 2. The different fascial planes in the neck by which infections may spread inferiorly into the mediastinum. The arrow demonstrates the standard surgical approach to the prevertebral fascia medial to the sternocleidomastoid muscle and carotid sheath and lateral to the strap muscles and thyroid gland.

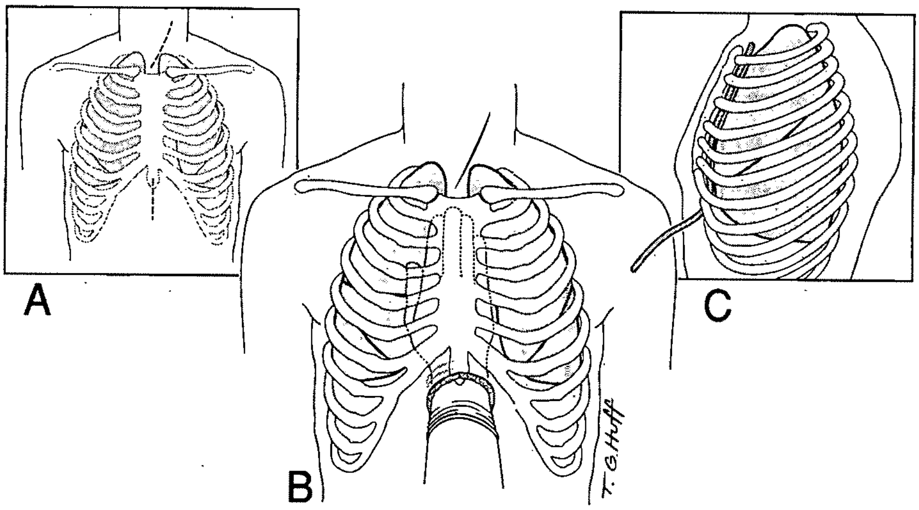

Fig 3. Subxiphoid drainage of the anterior mediastinum. Cervical and subxiphoid incisions (A) are followed by blunt manual retrosternal dissection (B), and then by placement of a large retrosternal drain brought out through the subxiphoid incision (C).

avenue for spread of infection from the oral cavity to the posterior mediastinum. Retropharyngeal abscesses from pharyngeal perforations, as can occur with traumatic endotracheal intubation [18, 27, 32], can rupture directly into the retrovisceral space, and from there spread to the posterior mediastinum. Because virulent pathogens can spread through fascial planes as well as along them, any cervical infection can potentially involve the entire mediastinum [8].

The CT scan is an extremely valuable tool in the management of cervical infections. The diagnosis of mediastinitis can be difficult, and plain roentgenograms are often nondiagnostic until sepsis has developed. The CT scan provides an early and accurate test for diagnosing the presence of mediastinitis, and is extremely useful in following the adequacy of surgical drainage.

In spite of the increased use of the CT scan, the mortality of DNM has remained essentially unchanged. In 1983, Estrera and associates [1] and Snow and co-workers [24] published comprehensive reviews recommending the use of CT scanning in all patients with serious cervical infections to determine the presence of mediastinitis. More importantly, these reports questioned the adequacy of standard transcervical mediastinal drainage for this condition. Estrera and associates state that "once the mediastinal necrotizing process is below the fourth thoracic vertebra posteriorly or the tracheal bifurcation, mediastinal drainage is best accomplished by a transthoracic approach." Improved results were noted with this more aggressive management. Nevertheless, transcervical drainage remains the most commonly employed approach to DNM. It is clear that the mortality for DNM remains prohibitively high because most patients do not receive adequate, early mediastinal drainage.

When DNM involves predominately the anterior mediastinum, adequate drainage may be achieved using a transcervical approach in combination with subxiphoid drainage, as demonstrated in our first patient (Fig 3). The anterior mediastinum may also be approached surgically by a parasternal extrapleural incision, as well as by a standard thoracotomy. Transpleural drainage has the disadvantage of causing pleural contamination, but should be used without hesitation if adequate drainage cannot be achieved by other means.

The adequacy of mediastinal drainage and the optimal surgical approach in patients with DNM is determined by the CT scan findings. If only the superior mediastinum is involved, and infection is contained above the level of the carina (the fourth thoracic vertebra), standard transcervical mediastinal drainage may be adequate. Patients with more extensive mediastinitis are best managed with a subxiphoid or chest incision, in addition to drainage of their cervical infections. A wait-and-see approach in these patients with extensive mediastinal infection is not appropriate. They are usually too sick to tolerate anything less than extensive, aggressive drainage with debridement of necrotic tissue.

Several other previously established tenets in the treatment of DNM should be reemphasized. First, tracheostomy remains an essential part of the management of the cervical infection, and should not be considered optional as has been suggested [33]. Because of the associated esophageal and cervical edema, it is nearly impossible to regain airway control in the circumstance of accidental endotracheal tube dislodgement after cervical drainage. Second, these patients are predisposed to other complications from their fulminant infection, including vessel erosion with exsanguination, aspiration, cranial nerve paralysis, intracranial suppuration, and necrotizing fasciitis [1, 34-36]. Finally, broad-spectrum aerobic and anaerobic antibiotic coverage should be initiated empirically as soon as possible; waiting for culture results with such aggressive infections is ill-advised.

Conclusion

Descending necrotizing mediastinitis remains a very aggressive form of mediastinitis, with a published mortality near 40%. Despite its origin in the oropharynx, this condition does not respond adequately to transcervical mediastinal drainage, which has become the standard therapy for localized acute mediastinitis associated with upper esophageal perforations. These two conditions, which require markedly different treatment, should not be confused. Computed tomographic scanning should be employed in cases of severe head and neck infections to establish the presence and extent of mediastinitis. Early, complete mediastinal drainage via a cervical incision and either a transthoracic or subxiphoid incision is essential to improve the chances for survival in these very ill patients.

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Improved Technique for Bilateral Lung Transplantation: Rationale and Initial Clinical Experience

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We previously described a technique for en bloc double-lung transplantation that was initially applied to select patients with cystic fibrosis and emphysema. This procedure is quite complex and associated with several limitations, including a substantial incidence of airway ischemia, postoperative myocardial depression, and cardiac denervation. To address these problems we have developed a simpler procedure for replacing both lungs. The operation is done through a transverse thoracosternotomy and involves sequential replacement of the two lungs. Positive features include separate bronchial anastomoses to reduce ischemic airway complications, elimination of the need for total cardiopulmonary bypass and a period of ischemic cardiac arrest, improved exposure to reduce intraoperative and postoperative hemorrhage, and maintenance of cardiac innervation. Additionally, the technique can be more easily mastered and widely applied. Details of the procedure and its initial clinical application in 3 patients having emphysema, cystic fibrosis, and bronchiolitis obliterans following previous double-lung transplantation, respectively, are described. All 3 patients recovered without complication. Postoperative function was excellent in spite of lung ischemic times ranging up to 9½ hours.

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After initial experience with single-lung transplantation in patients with end-stage pulmonary fibrosis [1, 2] we developed a procedure for en bloc double-lung transplantation for patients deemed unsuitable for unilateral lung transplantation [3]. Its initial application was in patients with obstructive lung disease, as it was felt that unilateral lung transplantation might result in overexpansion of the native lung, crowding of the transplanted lung, and ventilation-perfusion mismatch due to the overly compliant nature of the remaining emphysematous lung [4]. The double-lung transplant procedure, though complex, produced excellent results in the initial group of patients to whom it was applied [5]. Further experience, however, identified a number of limitations with this procedure, including a substantial incidence of tracheal anastomotic complications [6] and intraoperative hemorrhage when applying the procedure to patients with fused pleural space, such as in cystic fibrosis. Intraoperative and postoperative right ventricular dysfunction associated with the prolonged period of cardiopulmonary bypass, the need for a period of ischemic cardiac arrest, and the extensive cardiac manipulation during the extraction and implantation was often seen. Furthermore, long-term follow-up studies showed a high incidence of cardiac denervation associated with this procedure. This denervation is thought to be responsible for some limitation in maximum exercise tolerance, as has been described after combined heart-lung transplantation [7].

To address the issue of ischemic tracheal anastomotic problems, we pursued two avenues: exploration of techniques to restore bronchial artery circulation directly [8] and use of bilateral bronchial anastomoses instead of a tracheal anastomosis. Successful application of this latter technique has been reported by Noirclerc and associates [9]. To overcome the difficulties associated with en bloc double-lung transplantation, we developed a new procedure in the animal laboratory and refined it during human cadaver dissections. This bilateral lung transplantation procedure retains all of the advantages of the double-lung transplant procedure while eliminating many of its limitations. The technique and its successful application in 3 consecutive patients form the basis of this report.

Material and Methods

Surgical Technique

We have standardized the donor lung retrieval procedure that is used for single-lung transplantation, bilateral lung transplantation, and independent use of each lung in separate recipients. Pulmonary preservation consists of pulmonary artery perfusion with 3 L of cold modified Eurocollins solution. This is preceded by injection of 500 μg of prostaglandin E_1 into the pulmonary artery over a period of 60 seconds before inflow occlusion. The heart is first removed for cardiac transplantation using a previously described procedure that does not jeopardize either the use of the heart or use of the lungs [10]. After the heart

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has been removed, the lungs are removed en bloc along with the esophagus and the aorta. This technique was initially developed as part of a procedure to preserve the bronchial arteries and their origin from the aorta [8], and we have found that it expedites the safe removal of the lungs even though we are not currently using reimplantation of the bronchial arteries. The posterior mediastinal tissues are dissected to the spine inferiorly at the level of the diaphragm and superiorly at the midtracheal level above the aortic arch. The trachea and each end of the esophagus are closed with stapling devices. The entire double-lung–mediastinal bloc is dissected directly off the spine, placed in a plastic bag containing cold (4°C) normal saline solution, and transported surrounded by crushed ice in an ice chest. After arrival in the recipient operating room, the bloc is dissected on a back table while still immersed in sterile, cold saline solution. The esophagus and aorta are trimmed from the specimen, the pulmonary arteries are transected at their origin from the common pulmonary artery, and the posterior portion of the left atrium containing the four pulmonary veins is transected vertically in the midline to provide a satisfactory cuff of left atrium around the right and left pulmonary veins. Each bronchus is transected two cartilaginous rings above the origin of the upper lobe bronchus. The lungs are then wrapped in cold moist packs, immersed in cold saline solution, and partially covered with sterile crushed ice.

The recipient operation is performed with cardiopulmonary bypass available on a standby basis. When possible, an epidural catheter is placed preoperatively for management of postoperative pain. A left-sided double-lumen endotracheal tube is used for the procedure. The surgical procedure is initiated with an upper midline abdominal incision for mobilization of the omentum. Alternatively, omental preparation can be delayed until the end of the procedure if required for appropriate donor/recipient operative timing. The omental mobilization is carried out as for routine single-lung transplantation [11]. This usually requires no more than dissection of the omentum from the transverse colon. The omental pedicle is then divided longitudinally into two pedicles, carefully preserving adequate blood supply to each. A retrosternal tunnel is made for subsequent passage of the omental pedicles into the pleural spaces.

The excision and implantation of the lungs is carried out through bilateral anterior fourth or fifth interspace thoracotomy incisions extending from the midaxillary line to the sternal border. They are then joined by means of a transverse sternotomy. This so-called cross-bow approach provides superb access to the pleural spaces from the apex to the diaphragm, and to the posterior mediastinum on both sides, thereby greatly facilitating mobilization of the lungs and hilar structures.

With use of isolated left lung ventilation if necessary, the right lung is completely mobilized by dividing any adhesions to the chest wall, mediastinum, and diaphragm, and isolating the right pulmonary artery and pulmonary veins. To facilitate subsequent placement of the left atrial clamp for right lung implantation, the interatrial groove may be dissected at this point. The right lung is reinflated and the left lung is similarly mobilized, again using isolated right lung ventilation if required. Both lungs are then fully reexpanded.

The right lung transplantation is accomplished first. The Swan-Ganz catheter is positioned in the left pulmonary artery, with direct manipulation by the surgeon if necessary. The right lung is deflated while the patient is maintained on left lung ventilation. If this is not tolerated, partial bypass can be instituted at this point. The first branch of the pulmonary artery is divided between ligatures and the descending branch of the pulmonary artery is divided between ligatures or staple lines. Each pulmonary vein is divided between ligatures as peripherally as possible. The right main bronchus is transected just proximal to the right upper lobe and the right lung is removed.

The previously prepared right donor lung, wrapped in a cold, moist pack, is placed into the right chest and the bronchial anastomosis is performed using a running 4-0 absorbable monofilament suture for the membranous wall and interrupted 4-0 Vicryl (Ethicon, Somerville, NJ) for the cartilaginous portion. If the omentum has previously been prepared, an omental pedicle is wrapped around the bronchial anastomosis at this point. If not, it can be done at the end of the procedure.

The recipient pulmonary artery is then clamped centrally and the vessel trimmed to match the donor pulmonary artery. The anastomosis is performed with running 5-0 Prolene (Ethicon). A clamp is then placed on the recipient left atrium proximal to the pulmonary venous stumps. The ligatures are removed from the stumps of the pulmonary veins and an incision connects the openings of the two veins to form a suitable cuff of left atrium. The donor and recipient atrial cuffs are then anastomosed using running 4-0 Prolene suture interrupted at either end. Before the final suture is secured, the pulmonary artery clamp is released to clear air from the pulmonary vasculature until blood flushes through the venous suture line. The pulmonary artery clamp is replaced, the left atrial clamp is removed to allow backbleeding, and the atrial suture line is completed. The pulmonary artery clamp is then removed and the lung is inflated gently.

The left lung transplantation is now performed. The Swan-Ganz catheter is withdrawn into the common pulmonary artery and advanced into the right pulmonary artery. Because these initial 3 patients were severely disabled and hypoxemic preoperatively, and because the ischemic time for the right lung exceeded four hours, we chose to institute low-flow, partial cardiopulmonary bypass after right lung implantation. This avoided the necessity of imposing the full right-sided cardiac output immediately upon the reperfusing, transplanted right lung. For this technique the patient is heparinized (2 mg/kg) and cannulated for cardiopulmonary bypass. Standard ascending aortic cannulation for arterial inflow is easily accomplished through this exposure. A two-stage venous cannula is placed through the right atrial appendage for venous return. Partial cardiopulmonary bypass is used, allowing the right side of the heart to continue ejection. The bypass flow is adjusted to maintain the pulmonary systolic pressure below 30 mm Hg. The aver-

Table 1. Preoperative Patient Descriptors

Descriptor	Patient 1	Patient 2	Patient 3
Age (yr)	49	31	35
Sex	F	M	F
Diagnosis	Emphysema (α_1-antitrypsin deficiency)	Cystic fibrosis	Bronchiolitis obliterans (after DLTx)
Preop arterial blood gases			
pH	7.38	7.37	7.33
Oxygen tension (mm Hg)	73	61.0	64.8
Carbon dioxide tension (mm Hg)	57	70.2	41.0
Oxygen requirement (L/min)	6[a]	3	4[b]
Infected sputum production	++	++++	Purulent but no growth
Pulmonary function tests[c]			
FVC (L)	1.45 (41)	1.41 (26)	1.56 (39)
FEV_1 (L)	0.52 (19)	0.52 (12)	0.36 (11)
FVC/FEV_1	0.36	0.36	0.22
TLC (L)	5.01 (93)	5.95 (81)	2.93 (51)
RV (L)	2.99 (157)	4.26 (221)	1.37 (74)

[a] By oximizer. [b] Value on admission; this patient later became ventilator dependent. [c] Values in parentheses are percent predicted.

DLTx = double-lung transplantation; FEV_1 = forced expiratory volume in 1 second; FVC = forced vital capacity; RV = reserve volume; TLC = total lung capacity.

age flow rate for our 3 patients was 2 L/min. Although cardiopulmonary bypass was used in these patients, it should be considered elective, depending on the ischemic time and the hemodynamic status of the recipient.

The left lung is then excised and the donor lung implanted in a fashion similar to that described for the right lung, including omental wrap of the bronchus. As is normally done for single left lung transplantation, the ligamentum arteriosum is divided to facilitate proximal placement of the pulmonary artery clamp. After normal ventilation and perfusion are established to both lungs, cardiopulmonary bypass is discontinued and the patient is decannulated. Bronchoscopy is performed to clear the airways and to ensure that bronchial anastomoses are satisfactory. Because a double-lumen tube is in place, this must be done with a 3-mm bronchoscope. Two drainage tubes are placed in each thoracic cavity, the sternum is reapproximated with three sternal wires, and the thoracotomy incisions are closed in layers, including pericostal sutures.

At the termination of the operation, the double-lumen endotracheal tube is removed and the patient is reintubated with a standard endotracheal tube. Flexible bronchoscopy is then repeated to clear any secretions or blood before the patient is transferred to the intensive care unit.

Postoperative care is identical to that previously outlined by our group [12]. This includes hemodynamic monitoring, rigid control of immunosuppressive regimens, and aggressive pulmonary and physical rehabilitation. The epidural catheter remains in place until the patient is extubated and able to begin ambulation.

Description of 3 Recipients

Our initial experience with this bilateral lung transplant procedure in 3 consecutive patients is reported. The preoperative profile of the patients is outlined in Table 1. The patient population represented in this limited series spans the spectrum of indications for this procedure. The first patient had emphysema secondary to α_1-antitrypsin deficiency. Despite a prolonged period of pulmonary rehabilitation, her clinical course had continued to deteriorate as a result of recurrent bouts of infection. At the time of transplantation she was hospitalized with another bout of pulmonary sepsis, was bedridden, required 4 L of oxygen per minute via oximizer at rest, and had a carbon dioxide tension of 57 mm Hg.

The second patient was a 31-year-old man with cystic fibrosis who was seriously disabled and malnourished. Nutritional supplementation with enteral tube feeding before transplantation had assisted in increasing his weight from 41.4 to 51.3 kg. As with all patients with cystic fibrosis, his pulmonary sepsis excluded him from consideration of unilateral lung transplantation. His good cardiac function made heart-lung transplantation unnecessary.

The third patient had undergone standard double-lung transplantation 18 months before this procedure with an excellent functional result for the initial 15 months. Rapidly progressive obstructive lung disease then developed, diagnosed as bronchiolitis obliterans by both transbronchial and open lung biopsy. Her rapid deterioration progressed in spite of augmented immunosuppression including a course of OKT3. Over a 3-month period the forced expiratory volume in 1 second fell from 2.5 L to 300 mL. Her deterioration culminated in intubation, ventilatory support, and subsequent tracheostomy. Her copious sputum production precluded unilateral lung transplantation. At the time of operation, a portion of the omental pedicle used for the tracheal anastomosis of the original

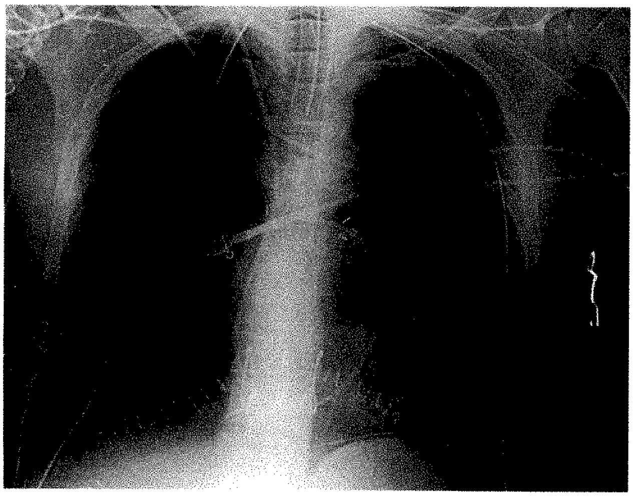

Fig 1. Immediate postoperative chest roentgenogram for patient 2.

double-lung transplant was retrieved and used to wrap the left bronchial anastomosis. An intercostal pedicle was used to wrap the right bronchial anastomosis. Before closure of the chest, the lungs were thought to be somewhat oversized for the chest. Because there was a large right middle lobe with complete fissures, a right middle lobectomy was performed to reduce the volume of transplanted lung.

Results

The operative and postoperative descriptors of these patients are displayed in Table 2. Of particular note is the ischemic time for the left lung, which ranged from 5 hours 50 minutes to 9 hours 15 minutes. All 3 patients had an immediate posttransplantation oxygen tension in excess of 400 mm Hg when measured on 100% oxygen. As seen in Table 2, the operative and postoperative courses were relatively uncomplicated. Specifically, all patients experienced stable perioperative hemodynamics and excellent lung function. Minimal postoperative hemorrhage was encountered despite the fact that 2 of the 3 patients (patients 2 and 3) were considered high risk for this complication. As seen in Table 2, all patients had satisfactory oxygenation on 30% oxygen on the first postoperative day. The first 2 patients were extubated on day 3 and 4, respectively, whereas patient 3, who had been on the ventilator with a tracheostomy before operation, was on a ventilator for 2 weeks because of poor respiratory mechanics.

The initial postoperative chest roentgenogram of patient 2 is shown in Figure 1. Late postoperative chest roentgenograms of patients 1 and 2 are shown in Figure 2.

Comments

We have been pleased with the results of unilateral single-lung transplantation for pulmonary fibrosis, and have recently extended its use to patients with primary pulmonary hypertension and to patients over the age of 50 years with chronic obstructive lung disease. For patients with cystic fibrosis or bilateral sepsis from other cause, we feel bilateral lung transplantation is the procedure of choice. Bilateral lung transplantation is also an option in younger patients with obstructive lung disease, especially when due to α_1-antitrypsin deficiency.

The bilateral lung transplant procedure requires two airway anastomoses. On the basis of our experience as well as that of others [9] with bilateral bronchial anastomosis, however, we feel this approach will result in a substantial reduction in airway complications when compared with the tracheal anastomosis as employed with the original double-lung procedure. The 6 bronchial anastomoses in our 3 patients healed without complication. All are wrapped with omentum except for an intercostal muscle flap used on the right bronchus in the patient who underwent a redo double-lung replacement. Our experience with single-lung transplantation suggests that even if complications do occur with the bronchial anastomosis, disruptions are contained by the omental wrap and usually heal satisfactorily with time. The experience of one of us (J.D.C.) includes 40 unilateral or bilateral bronchial anastomoses for single and bilateral lung transplantation. In this group there have been two deaths attributable to bronchial complications. Partial dehiscence requiring temporary or permanent replacement of a silicone stent (Hood Laboratories, Pembrooke, MA) developed in 4 other patients.

The bilateral lung transplant procedure allows replacement of both lungs without dissection of the distal trachea, subcarinal region, and posterior mediastinum. This is a particular advantage in patients with cystic fibrosis in whom bulky, vascular mediastinal lymphadenopathy, combined with lack of access to the posterior mediastinum, may make control of postoperative hemorrhage exceedingly difficult through the midline sternotomy approach as used for the double-lung procedure.

Cardiopulmonary bypass was used in all 3 of our initial patients because of the specifics of these individual cases. As previously noted, the procedure should be done with cardiopulmonary bypass available on a standby basis. Its use during replacement of the left lung should be considered optional.

The absence of any induced cardiac ischemic period, as required for the left atrial anastomosis of the double-lung procedure, also weighs heavily in favor of the bilateral single-lung transplant approach. Patients with mild to moderate cardiac dysfunction that would have excluded them from consideration of a double-lung transplantation may tolerate this simplified procedure with minimal difficulty. The operative course of our patients was notably free of hemodynamic instability in contrast to our experience with the previously employed double-lung transplant procedure. This simplified the postoperative care and permitted earlier institution of pulmonary and physical rehabilitation with resulting earlier extubation and ambulation.

Limitations

Despite our enthusiasm for the simplicity and applicability of this operative procedure, this report must be con-

Table 2. Operative and Postoperative Descriptors

Descriptor	Patient 1	Patient 2	Patient 3
Donor lung ischemic time			
Right	4 h 17 min	5 h 2 min	6 h
Left	5 h 50 min	7 h 15 min	9 h 15 min
Time on CPB	1 h 32 min	1 h 58 min	6 h 5 min
Average CPB flow rate (L/min)	1.9	1.7	2.0
Postop inotropes required	None	None	Amrinone, 5 μg/kg/min
Immediate postop lung perfusion scan			
Right	59%	55%	80%[a]
Left	41%	45%	20%[a]
Late postop lung perfusion scan			
Time (wk)	7	4	4
Right	52%	45%	58%
Left	48%	55%	42%
Early arterial blood gases[b]			
Oxygen tension (mm Hg)[c]	122	79	118
pH	7.44	7.47	7.41
Carbon dioxide tension (mm Hg)	36	40	36
HCO_3 (mEq/L)	25	30	24
Discharge arterial blood gases[d]			
Day	24	18	29
Oxygen tension (mm Hg)	110	111	97
Carbon dioxide tension (mm Hg)	47	37	38
pH	7.51	7.4	7.36
HCO_3 (mEq/L)	37	24	22
Extubation	POD 3	POD 4	Tracheostomy preop; off ventilator POD 14
Discharge from ICU	POD 6	POD 5	POD 15
Discharge home	POD 24	POD 19	POD 29
Discharge pulmonary function tests[e]			
FVC (L)	1.90 (53)	3.02 (55)	1.55 (39)
FEV_1 (L)	1.88 (69)	2.91 (68)	1.27 (40)
FEV_1/FVC	0.98	0.96	0.82
TLC (L)	4.43 (82)	6.73 (92)	3.80 (67)
RV (L)	2.53 (132)	3.71 (192)	2.25 (121)
Latest pulmonary function tests and arterial blood gases[e]			
Time (wk)	9	16	9
FVC (L)	2.49 (70)	3.89 (70)	1.83 (46)
FEV_1 (L)	2.42 (89)	3.80 (88)	1.47 (47)
FEV_1/FVC	0.97	0.97	0.80
TLC (L)	4.89 (91)	6.58 (90)	3.43 (60)
RV (L)	2.40 (126)	2.69 (139)	1.60 (86)
pH	7.47	7.43	7.42
Oxygen tension (mm Hg)[d]	86.9	86.5	7.91
Carbon dioxide tension (mm Hg)[d]	37.8	39.3	42
Airway healing	Satisfactory	Satisfactory	Satisfactory

[a] These values are for the day of operation; on postop day 1, the values were: right, 50%; left, 50%. [b] Postop day 1. [c] On 30% inspired oxygen fraction. [d] On room air. [e] Values in parentheses are percent predicted.

CPB = cardiopulmonary bypass; FEV_1 = forced expiratory volume in 1 second; FVC = forced vital capacity; ICU = intensive care unit; POD = postoperative day; RV = reserve volume; TLC = total lung capacity.

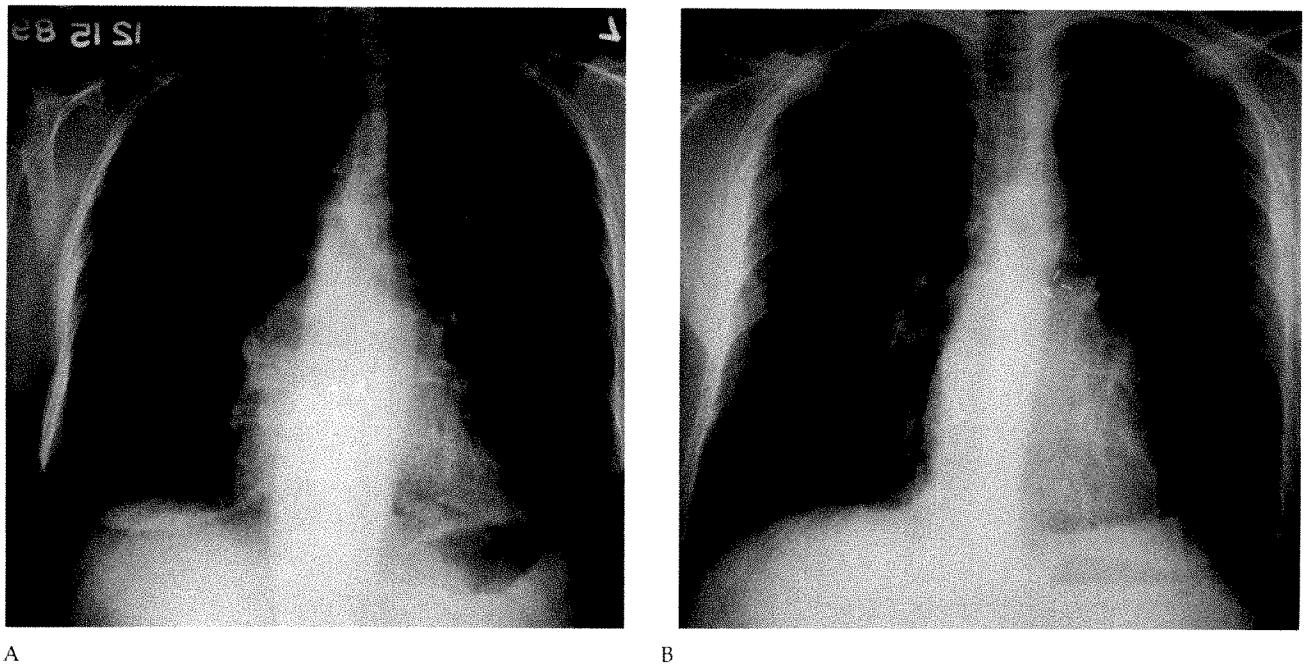

Fig 2. (A) Chest roentgenogram of patient 1 seven weeks after transplantation. (B) Chest roentgenogram of patient 2 six weeks after transplantation.

sidered preliminary and further experience will be required before it can be unconditionally recommended. We were initially concerned that the bilateral thoracosternotomy incision might result in serious postoperative morbidity and be especially poorly tolerated by the group of chronically ill, severely disabled patients requiring this procedure. Such has not proved to be the case and early extubation and mobilization has been possible, aided, at least in part, by the use of an epidural catheter for analgesia.

Another potential limitation of this procedure may be the inevitable lengthening of the ischemic time for the second lung. In these 3 cases, postoperative gas exchange was excellent in spite of ischemic times ranging up to 9½ hours. To further assess the effects of the longer ischemic time on the second lung, we obtained quantitative lung perfusion scans immediately after the procedure in all patients (see Table 2). In the first 2 patients, having left lung ischemic times of 5 hours 50 minutes and 7 hours 15 minutes, respectively, the quantitative perfusion scan showed a normal distribution. In the third patient, with a 6-hour ischemic time for the right lung and a 9-hour 15-minute ischemic time for the left lung, the initial quantitative perfusion scan demonstrated 80% of perfusion to the right lung and 20% to the left lung, which we interpret as due to ischemic injury to the left lung. On the following day, however, quantitative perfusion scan showed 50% to each lung. This is in keeping with our previously reported experience in animals, showing initial dysfunction and subsequent recovery after 12 hours of ischemia [13].

Conclusion

Because of the advantages of this bilateral transplant procedure and our favorable experience to date, we no longer perform en bloc double-lung transplantation. We employ the bilateral lung transplant procedure for all patients with cystic fibrosis, and for patients with end-stage chronic obstructive lung disease under the age of 50 years. We still use single-lung transplantation for patients with pulmonary fibrosis, select patients with pulmonary hypertension, and patients over the age of 50 years with emphysema.

Addendum

We have now performed an additional six bilateral lung transplantations, three of which were performed without the need for cardiopulmonary bypass. All patients were extubated and had normal blood gases on room air by the third postoperative day. Healing of the airway anastomoses has been normal and there has been no serious morbidity and no mortality in this overall series of 9 patients.

This work was supported by the National Cystic Fibrosis Foundation.

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Notice From the American Board of Thoracic Surgery

The American Board of Thoracic Surgery began its recertification process in 1984. Diplomates interested in participating in this examination should maintain a documented list of the operations they performed during the year prior to application for recertification. They should also keep a record of their attendance at thoracic surgical meetings, and other continuing medical education activities, for the 2 years prior to application. A minimum of 100 hours of approved CME activity is required.

In place of a cognitive examination, candidates for recertification will be required to complete both the general thoracic and cardiac portions of the SESATS III syllabus (Self-Education/Self-Assessment in Thoracic Surgery). It is not necessary for candidates to purchase SESATS III booklets prior to applying for recertification. SESATS III booklets will be forwarded to candidates after their applications have been accepted.

Diplomates whose 10-year certificates will expire in 1992 may begin the recertification process in 1990. This new certificate will be dated 10 years from the time of expiration of the original certificate. Recertification is also open to any diplomate with an unlimited certificate and will in no way affect the validity of the original certificate.

The deadline for submission of applications is July 1, 1990. A recertification brochure outlining the rules and requirements for recertification in thoracic surgery is available upon request from the American Board of Thoracic Surgery, One Rotary Center, Suite 803, Evanston, IL 60201.

Effect of Storage at 4°C in a Nutrient Medium on Antigenic Properties of Rat Aortic Valve Allografts

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The effects of prolonged storage at 4°C in nutrient medium on the antigenic properties of aortic valve allografts are unknown. Lewis rats received heterotopic aortic valve allografts from Brown Norway donors. Valves were transplanted immediately after harvest (fresh), or after antibiotic sterilization and storage in a nutrient medium at 4°C for 3, 7, 14, or 21 days. Additional rats underwent sham laparotomy (sham). All recipient rats received Brown Norway skin grafts 3 weeks after valve transplant or sham procedure. Time to skin graft rejection for all groups was as follows: fresh ($n = 10$), 4.5 ± 0.9 days; sham ($n = 10$), 7.1 ± 0.3 days; 3-day ($n = 10$), 4.9 ± 0.3 days; 7-day ($n = 10$), 5.2 ± 0.4 days; 14-day ($n = 10$), 6.1 ± 0.7 days; and 21-day ($n = 10$), 6.0 ± 0.6 days. Significant differences existed between the sham group and each of the transplanted groups. No significant differences existed between the fresh group and either the 3-day or 7-day groups. The difference between the fresh group and the 14-day group approached significance ($0.05 < p < 0.10$), and the difference between the fresh and 21-day groups was significant ($p < 0.05$). Additional valves not used for transplantation were studied with scanning electron microscopy. The valves preserved in nutrient medium exhibited a progressive loss of endothelium as compared with fresh valves. Storage of aortic valve allografts in a nutrient medium at 4°C is associated with a progressive attenuation of antigenic response. The loss of antigenicity appears to plateau, however, and the grafts remain immunogenic even after 3 weeks. Loss of endothelial integrity may explain the change in immunological effect.

(Ann Thorac Surg 1990;49:792–6)

Since their introduction in 1962, aortic valve allografts have gradually become established as alternatives to mechanical or porcine prostheses for treatment of aortic valve disease. The excellent immediate results obtained by a number of centers have defined the necessary operative technique to permit more widespread use. With increasing application of allografts, consideration of factors that affect long-term survival of both graft and patient has become necessary. The importance of the immunological properties of valve allografts is uncertain. Although no evidence shows that ABO blood group mismatching adversely affects graft survival [1, 2], some cardiac surgeons recommend that donor–recipient matching should be performed if possible [3].

This study was designed to evaluate one method of preservation used clinically, consisting of antibiotic sterilization and storage in a nutrient medium. This method was examined in a rat model using heterotopic valve transplantation. Second-set skin graft rejection was measured to determine the effect of prolonged storage before transplantation on the immunological properties of the valve graft. Electron microscopic examination of the valves was performed to define the ultrastructural changes that may correlate with changes in antigenic expression.

Material and Methods

Inbred male rats weighing 250 to 300 g obtained from Harlan Sprague-Dawley (Indianapolis, IN) underwent general anesthesia with 3.6% chloral hydrate solution (1 mL/100 g of body weight) administered by intraperitoneal injection. Under clean conditions, the heart and ascending aorta were excised and rinsed in 0.9% saline solution. The aortic valve with attached ascending aorta, anterior mitral leaflet, and a small portion of left ventricular outflow tract was dissected. Valves were immediately transplanted or were placed in cold RPMI 1640 with 10% fetal bovine serum and the following antibiotics: gentamicin (160 mg/L), piperacillin (1,000 mg/L), oxacillin (50 mg/L), metronidazole (200 mg/L), and amphotericin B (100 mg/L). Warm ischemic time was less than ten minutes. After 24 hours in the antibiotics, the valves were rinsed and stored at 4°C in fresh RPMI 1640 with 10% fetal bovine serum for 3, 7, 14, or 21 days before transplantation.

Valve transplantation was performed under clean conditions by the method of Yankah and associates [3] using a ×14 operating microscope. After induction of general anesthesia with chloral hydrate, recipient rats underwent laparotomy and dissection of the abdominal aorta. The allograft was prepared for implantation by suture ligation of the coronary arteries. The recipient aorta was occluded proximally and distally and divided. An end-to-end anastomosis between the proximal aorta and the proximal (ventricular) end of the valve graft was performed using 8-0 polypropylene suture. The anterior leaflet of the aortic valve was incorporated into the suture line to prevent

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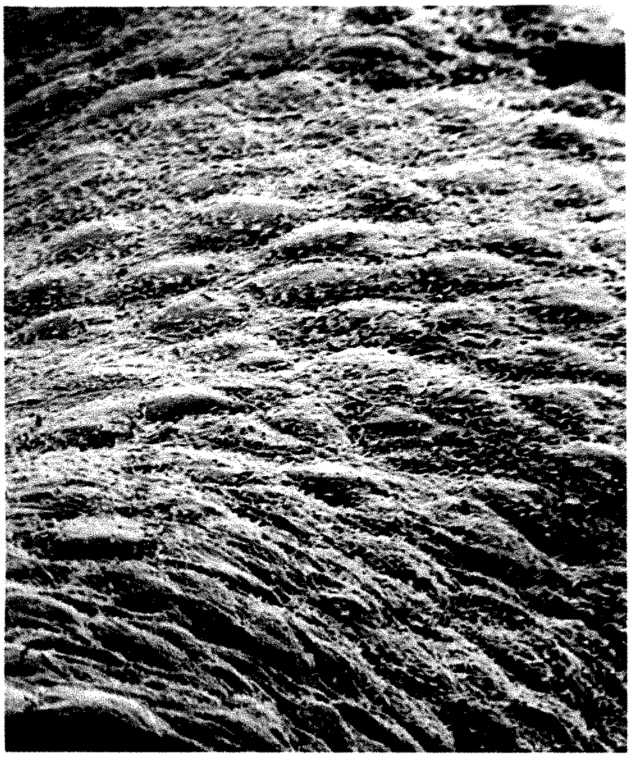

Fig 1. Scanning electron micrograph of rat aortic valve leaflet immediately after harvest (×1,500 before 32% reduction). The endothelium is intact.

valve competence, which may predispose to thrombosis. The distal (aortic) end of the graft was similarly anastomosed to the recipient distal aorta. Aortic occlusion time was less than 30 minutes. The abdomen was closed and the animals were allowed to recover. Sham-operated animals underwent laparotomy and aortic dissection alone. All valve recipients and sham-operated animals were of the Lewis strain. Valve donors were of the Brown Norway strain (strongly allogeneic, RT1 and non-RT1 incompatible).

Three weeks after transplantation or sham operation, the animals received a full-thickness skin graft from the ear of a Brown Norway rat applied to the recipient's lateral thoracic wall. The rats were inspected daily beginning three days after skin grafting. Skin graft rejection was defined by standard criteria [4].

In all, 60 recipient Lewis rats were studied: 10 fresh valve recipients; 10 each receiving grafts preserved 3, 7, 14, or 21 days; and 10 sham-operated controls.

The time to skin graft rejection was recorded, and the mean and standard deviation for each group was calculated. Data were analyzed using a one-way analysis of variance. When significant differences were found by analysis of variance, multiple *t* tests were performed to detect differences between each of the stored groups and the sham and fresh controls. Bonferroni's inequality for multiple comparisons (eight comparisons) was applied [5].

Additional valves not used for transplantation were examined after fixation in 2.5% glutaraldehyde buffered with 0.1 mol/L of cacodylate to pH 7.4. Tissues were dehydrated by graded ethyl alcohol solutions, subjected to critical point drying, and coated with carbon/platinum. The valve leaflets were examined for endothelial structure using a Hitachi S 570 and H 4000 scanning electron microscope. Three fresh valves and three from each storage period were studied.

Animals received humane care in compliance with the "Principles of Laboratory Animal Care" formulated by the National Society for Medical Research and the "Guide for the Care and Use of Laboratory Animals" prepared by the National Academy of Sciences and published by the National Institutes of Health (NIH publication No. 85-23, revised 1985).

Results

Duration of skin graft survival is shown in Table 1. With increasing duration of storage before transplantation, there was an increase in skin graft survival. Significant differences existed between the sham controls and all groups of stored valves. However, the 21-day group exhibited significantly prolonged survival as compared with the fresh group. In addition, the 14-day group approached but did not reach a significant difference as compared with the fresh group.

Scanning electron microscopy of the leaflets of fresh valves showed an intact layer of endothelial cells, with no evidence of structural damage (Fig 1). The valves preserved for three days exhibited some loss of endothelial cells (Fig 2). Large areas of denudation and peeling were apparent after seven days of storage (Fig 3). After 14 or 21 days of storage (Figs 4, 5), no endothelial cells could be identified.

Comment

Despite the long experience with aortic valve allografts and recent increase in their application, a number of basic questions remain regarding the optimum method of harvest, processing, and storage of these materials. Clinical studies are hampered by evolution in techniques over time, including such important factors as method of

Table 1. Duration of Skin Graft Survival

Valve treatment[a]	Duration (days)[b]	*p* Value Versus Sham	*p* Value Versus Fresh
Fresh	4.5 ± 0.9	. . .	. . .
Sham	7.1 ± 0.3	. . .	. . .
Stored at 4°C			
3 days	4.9 ± 0.3	<0.05	NS
7 days	5.2 ± 0.4	<0.05	NS
14 days	6.1 ± 0.7	<0.05	$0.05 < p < 0.10$
21 days	6.0 ± 0.6	<0.05	<0.05

[a] n = 10 for each group. [b] Values are shown as mean ± standard deviation.

NS = not significant.

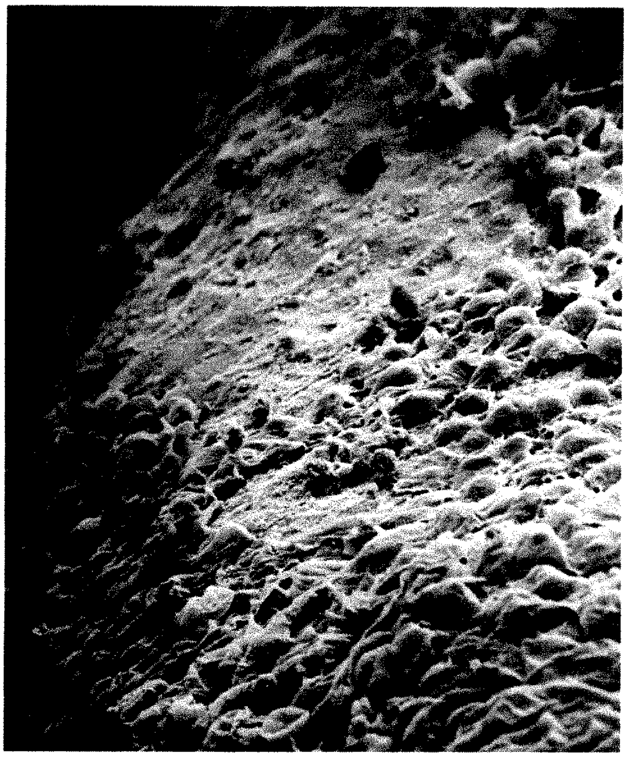

Fig 2. Scanning electron micrograph of rat aortic valve leaflet after three days of storage in nutrient medium at 4°C (×600 before 32% reduction). Patchy areas of endothelial loss are evident.

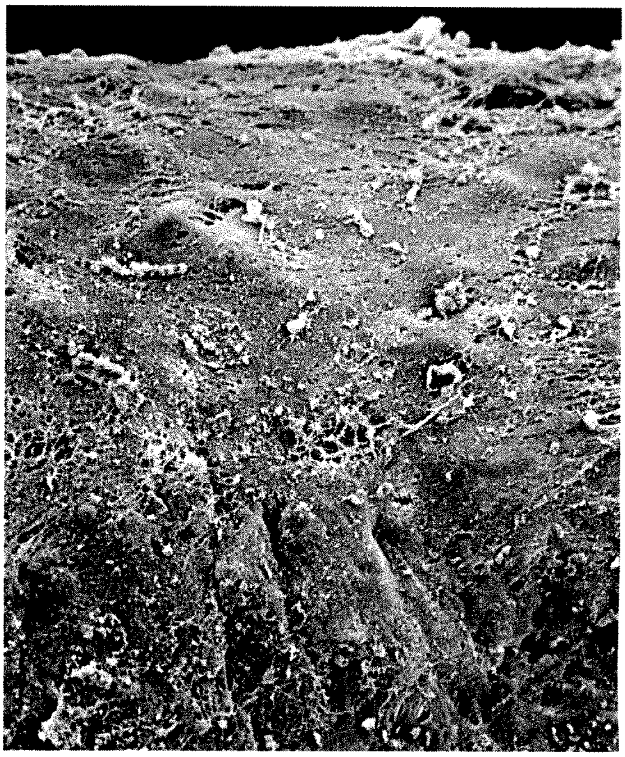

Fig 4. Scanning electron micrograph of rat aortic valve leaflet after 14 days of storage in nutrient medium at 4°C (×1,100 before 32% reduction). No identifiable endothelial cells are apparent.

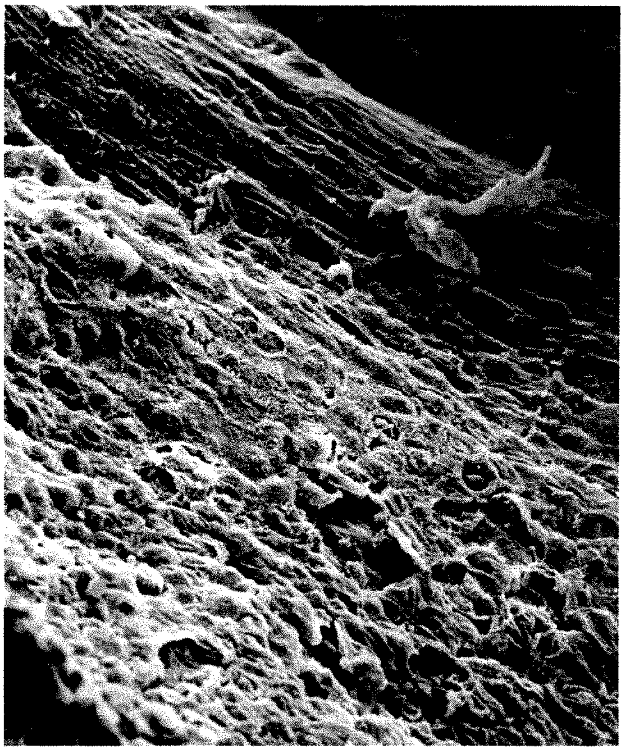

Fig 3. Scanning electron micrograph of rat aortic valve leaflet after seven days of storage in nutrient medium at 4°C (×600 before 32% reduction). Large areas of endothelial denudation are evident.

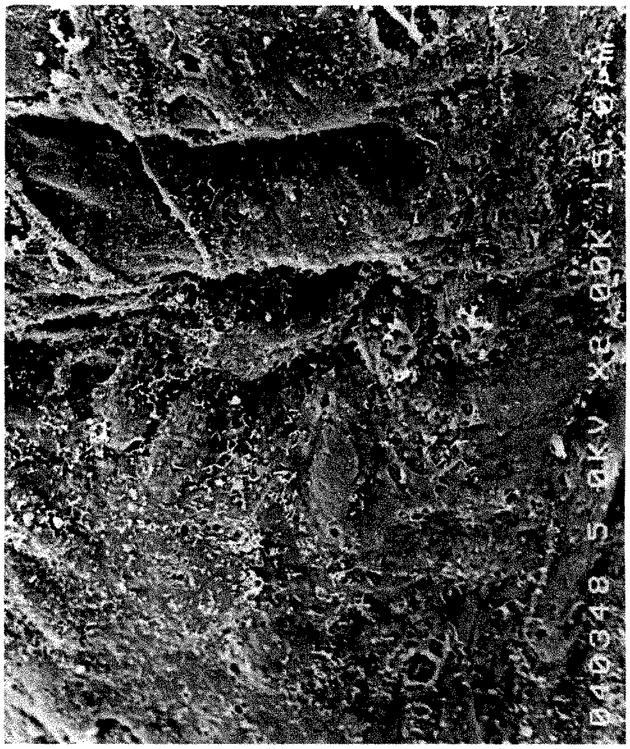

Fig 5. Scanning electron micrograph of rat aortic valve leaflet after 21 days of storage in nutrient medium at 4°C (×2,000 before 32% reduction). As in the 14-day group, there is complete loss of endothelium.

sterilization, use of nutrient medium, and depth of hypothermia employed. Most critical is the recognition that patient factors, perhaps more than valve factors, largely determine long-term valve performance [6]. Therefore, an animal model may be necessary to evaluate some of the vital questions regarding valve allografts.

The clinical significance of immunological differences between valve donor and recipient is unclear. The few large series of patients with allograft valves have failed to discern any demonstrable immunological reaction [1, 7]. Nevertheless, the hope that allograft valves would prove "immunologically privileged" is probably unjustified. It has been stated that only fresh allografts are capable of provoking an immune response [8]. This assertion makes ambiguous usage of the word "fresh," which has come to include both valves implanted immediately after harvest and those stored for varying periods of time. Nevertheless, we [9] and other investigators [10] have shown that modern techniques of cryopreservation result in maintenance of antigenic properties indistinguishable from those of untreated tissue. The present study showed that antigenicity of rat aortic valves is preserved even after 3-week storage at 4°C. These data appear to demonstrate a progressive attenuation of antigenicity, however, that reaches a plateau but is not eliminated.

The explanation for the decrease in immunological potency may be found in the structural changes of the valves. Yankah and co-workers [11] used antibiotic sterilization and storage in a nutrient medium at 4°C to study rat aortic valve endothelium. They demonstrated that endothelial viability, assessed by Alcian blue staining, decreases rapidly within the first 48 hours after valve allograft harvest, and decreases further, although less rapidly, through the subsequent three to seven days. The loss of endothelium with prolonged storage may result in a lower antigen burden presented to the recipient. Because endothelium strongly expresses both class I and class II antigens and is the most immunostimulatory component of vascular structures [12], the loss of these cells may have a disproportionately great influence on valve antigenicity. The present investigation shows that despite extensive loss of endothelium by 21 days, the valves continued to produce acceleration of skin graft rejection as compared with the sham controls. This recipient sensitization may persist because the fibroblasts or other components of the valve also maintain important immunological properties. Ventricular muscle, which was included in the grafts, plays an important part in the antigenic effects of aortic valve allografts [13].

The ultimate implications of endothelial loss for long-term allograft performance are unknown. Vascular endothelium synthesizes components of connective tissue, prevents platelet adherence, elaborates prostacyclin, accelerates thrombin inactivation, and contributes to fibrinolysis [14]. Loss of these properties may prove deleterious to graft longevity. Reduction of the immunological burden may, however, have an offsetting benefit that enhances graft performance.

The method of valve storage used in this study was analogous to that used clinically in some centers [15, 16]. The antibiotic sterilization used was free of drugs known to have an adverse effect on collagen cross-linking or ground substance [16]. A nutrient medium was used because of evidence that such a solution enhances cellular viability [17]. The presence of fetal calf serum has been challenged as a factor that may adversely affect allograft valve longevity [18]. Many investigators [19, 20] are convinced, however, that optimum cellular viability is a critical determinant of long-term allograft performance. We believe that the benefits of nutrient medium on cellular viability justify use of such solutions in valve preservation.

"Viability" is unlikely to be an all-or-none property [21, 22]. Similarly, "antigenicity" is a complex characteristic determined by degree of histoincompatibility, cellular viability, surface antigen expression, metabolic activity, number of cells, and location of cells [23]. Both these properties may therefore be somewhat separable, mutable, and gradational. Perhaps selective enhancement of one quality can be achieved without adversely affecting the other, thereby improving valve performance. To accomplish such selective improvement in valve characteristics, we must first define these qualities. Although the ultimate test of valve storage techniques is the clinical performance in adequate numbers of patients, use of this animal model may facilitate the necessary comparisons.

The results of this study suggest that aortic valve allografts stored at 4°C in a nutrient medium maintain their antigenic potential despite storage for the longest period of time consistent with clinical usability [24]. They further suggest that a significant quantitative decrease occurs in the immunological stimulus with long periods of storage, and that this may be attributable to a loss of endothelial cells. Whether this loss of endothelium has a beneficial, detrimental, or neutral effect on long-term performance of the valve is unknown.

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Modified Blalock-Taussig Shunts: Results in Infants Less Than 3 Months of Age

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The optimal procedure for shunting palliation in cyanotic infants remains to be determined. Sixty-two infants less than 3 months of age underwent 63 modified Blalock-Taussig shunts. Their age range at operation was 1 to 84 days (mean, 16 ± 20 days). Shunts were constructed using 5-mm polytetrafluoroethylene tubes in 20 patients and 4-mm polytetrafluoroethylene grafts in 43 patients. There were 13 early deaths (21%; CL, 15% to 27%) of which three deaths (5%; confidence limits, 2% to 9%) were shunt related. The survivors were followed up from 6 to 53 months (mean, 29 ± 12.5 months). Shunt failure (occlusion, inadequate palliation) occurred in 27 patients. The overall probability rate of adequate shunt function was 58% ± 8% at 2 years. Univariate and multivariate analyses showed that the size of the graft was a risk factor of shunt failure. Severe distortion of the pulmonary arterial branch was noted in 12 patients. The inferences are: (1) modified Blalock-Taussig shunts provide satisfactory early palliation but late shunt failure is frequent; (2) similar results should be obtained with other shunting procedures; and (3) the optimal procedure should be selected for each cyanotic infant on an individual basis.

(Ann Thorac Surg 1990;49:797–801)

Recent advances in pediatric cardiac surgery allow early primary complete repair in many patients with congenital heart malformations and decreased pulmonary blood flow. However, a substantial number of patients still require a palliative procedure to increase pulmonary blood flow during infancy.

The Potts and Waterston shunts have been strongly advocated and then abandoned in most centers. The classic Blalock-Taussig shunt remains the shunt of choice in children, but it is questioned in infants because of a high rate of inadequate palliation. The modified Blalock-Taussig shunt (MBTS) using a polytetrafluoroethylene conduit was first designed for patients in whom a classic Blalock-Taussig shunt could not be carried out. Satisfactory results were obtained, and many centers considered the MBTS the procedure of choice, particularly in infants. The MBTS offers several advantages: (1) high early patency rate, (2) regulation of the shunt flow by the size of the systemic artery, (3) preservation of the subclavian artery, and (4) ease of the surgical procedure.

Early results with the MBTS have been widely reported. However, reports on long-term results, especially in infants, remain scarce. Our experience with the MBTS in infants less than 3 months of age is reported in an attempt to identify the predicting factors of adequate shunt function.

Material and Methods

Patient Population

Sixty-two infants (age, <3 months) underwent 63 MBTS procedures between January 1, 1984, and December 31, 1987. The mean age (± standard deviation) was 16 ± 20 days (range, 1 to 84 days). Fifty-six patients (89%) were neonates (<1 month of age); 29 (46%) were newborns (<1 week of age). The mean weight (± standard deviation) was 3.2 ± 0.5 kg (range, 2.1 to 4.9 kg).

The diagnoses were grouped into four categories (Table 1): group 1, tetralogy complex (15 patients); group 2, transposition complex (11 patients); group 3, univentricular heart (13 patients); and group 4, pulmonary atresia or critical stenosis with intact ventricular septum (24 patients). The pulmonary artery was atretic in 40 patients (63.5%). The size of the pulmonary arterial branch, as assessed at operation, was 3 mm in 2 patients, 4 mm in 16 patients, 5 mm in 15 patients, 6 mm in 13 patients, 7 mm in 14 patients, and 8 mm in 2 patients.

Surgical Technique

The shunts were constructed by interposing a conduit between the subclavian artery and the ipsilateral pulmonary arterial branch. Systemic and pulmonary anastomoses were done end to side using a running polypropylene suture. Sixty-three thin-walled polytetrafluoroethylene tubes (Gore-Tex, William Gore, Inc, Flagstaff, AZ) (5 mm in 20 shunts, 4 mm in 43 shunts) were used.

Fifty-six shunts were performed on the right side and seven, on the left side. The procedure was done through a standard lateral thoracotomy except in 13 infants with pulmonary atresia and intact ventricular septum in whom concomitant placement of a right-sided MBTS and closed pulmonary valvotomy were performed through a median sternotomy. Heparin was not administered before or during operation but was given at a low dosage (1 mg/kg/day) during the first three to five postoperative days and then replaced by long-term aspirin therapy.

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Table 1. Diagnoses of Intracardiac Anomalies

No. of Patients	With Pulmonary Stenosis	With Pulmonary Atresia	Total
Tetralogy complex	4	11	15
Transposition complex	7	4	11
Univentricular heart (includes tricuspid atresia)	8	5	13
Pulmonary atresia or critical stenosis and intact ventricular septum	4	20	24

Shunt Patency, Adequacy of Palliation, and Follow-up

Shunt patency was determined using a combination of clinical findings, two-dimensional Doppler echocardiography, and cardiac catheterization. Shunt failure was defined by one of the following: occlusion, reappearance of cyanosis with progressive decrease in oxygen tension, or need for a second shunt or complete repair (nonelective). Length of time from anastomosis to failure was noted.

Follow-up data (100% complete) were obtained during the 3-month interval from May to July 1988. Children not followed up at this institution were contacted through referring physicians.

Pulmonary Artery Distortion

Postoperative angiography was available in 34 patients. Cardiac angiography was performed for shunt failure (18 patients) or for scheduled elective operation (16 patients). Pulmonary artery distortion was defined as a stenosis severe enough to require correction at the time of definitive intracardiac repair.

Statistical Analysis

Continuous data are presented as means ± 1 standard deviation and crude ratios with 70% confidence limits (CL). Univariate and multivariate analyses were used to evaluate the adequacy of palliation and to assess the importance of the following risk factors: age, weight, presence of pulmonary atresia, diagnostic groups, size of the pulmonary arterial branch, and size of graft. In univariate analysis, product-limit methods (Kaplan and Meier) and log-rank test (BMDP1L) [1] for comparison of survival curves were used to evaluate the duration of adequate shunt function. The multivariate analysis used the stepwise Cox regression model (BMDP2L) [1]. Statistical significance was established at $p = 0.05$.

Results

Early Mortality (Within 30 Days)

Two intraoperative and 11 postoperative deaths yielded an overall early mortality rate of 21% (CL, 15% to 27%). All deaths but two occurred in neonates. Three early deaths (5%; CL, 2% to 9%) were shunt related. Two shunts were patent but inadequate; the patients died despite an urgent second shunt procedure. One infant had early heart failure, probably related to excessive pulmonary blood flow, and died despite reduction of the shunt. The other causes of death were: low cardiac output (3 patients), sepsis (2), sudden death (2), and surgical mismanagement (1 patient).

Early Shunt Failure (Within 30 Days)

Early shunt failure occurred in 5 patients (8%; CL, 4% to 13%) (Table 2). Two shunts were occluded, and three were patent but inadequate. Four infants underwent a second shunt (three central shunts and one contralateral MBTS); 2 patients died during reoperation. One patient underwent a successful intracardiac repair.

Late Results

Twenty-two shunt failures occurred during follow-up. Eleven patients underwent a second shunt procedure, and 8 had nonelective intracardiac repair. One child, who lived in a distant foreign country, died before reoperation could be performed. Two patients with pulmonary atresia and intact ventricular septum who had undergone concomitant placement of an MBTS and pulmonary valvotomy had shunt thrombosis but did not require reoperation owing to an adequate valvotomy.

The overall adequate palliation rates estimated by product limit methods were 85% ± 5% at 1 year, 58% ± 8% at 2 years, and 21% ± 8% at 3 years (Fig 1). Univariate and multivariate analyses showed that the size of graft was a significant risk factor of shunt failure (Tables 3, 4). The probability rate of adequate palliation was significantly lower with a 4-mm polytetrafluoroethylene conduit (Fig 2).

Distortion of the Pulmonary Arterial Branches

Severe distortion of the pulmonary arterial branch was observed in 12 patients (Table 5). Among 41 MBTSs functioning more than 3 months, 10 of the 20 failed shunts showed distortion of the pulmonary artery at the site of the anastomosis.

Table 2. Early Shunt Failures

Intracardiac Anomaly	Age (days)	Graft Size (mm)	Subsequent Operation	Result
Critical pulmonary stenosis and intact ventricular septum	20	4	Complete repair	Survived
Tetralogy with PA	9	4	Contralateral shunt	Survived
Tricuspid atresia	7	4	Central shunt	Died
Tricuspid atresia	13	4	Central shunt	Survived
Tetralogy with PA and hypoplasia of pulmonary arterial tree	5	4	Central shunt	Died

PA = pulmonary atresia.

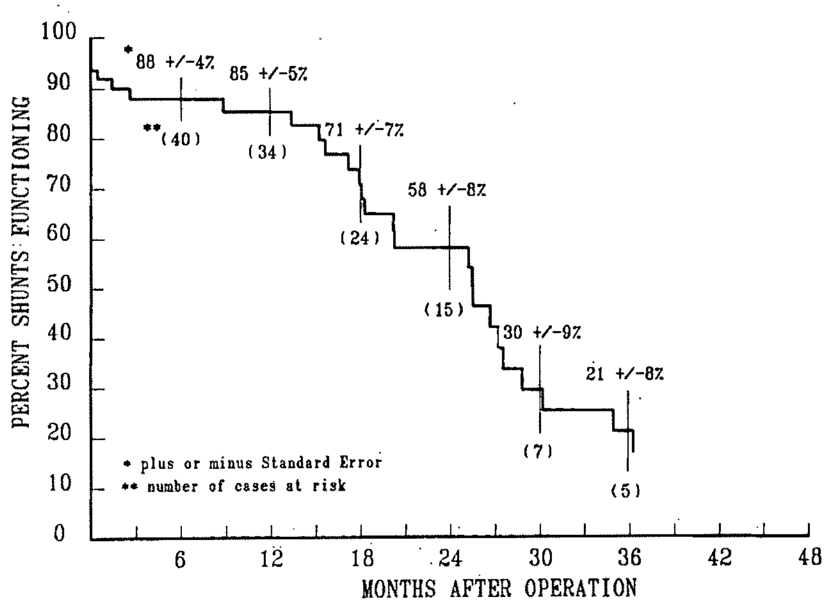

Fig 1. Actuarial probability of adequate shunt function for all shunts.

Comment

Shunting procedures are indicated for critically ill cyanotic newborns or infants with complex malformations that are not amenable to primary complete repair [2]. The ideal shunt should (1) provide adequate pulmonary blood flow, (2) allow shunt flow to increase with growth, (3) be reliable in its early and late flow characteristics, (4) provide bilateral pulmonary arterial flow, (5) avoid distortion of the pulmonary arterial anatomy, and (6) be simple to construct and to take down [3]. There is wide agreement to consider the MBTS [4] the procedure of choice [2, 3, 5–8].

Table 3. Risk Factors of Shunt Failure (Univariate Analysis)

Risk Factor	Number of Shunts	Observed Failures	*p* Value
Age			0.88
≤30 days	55	24	
>30 days	8	3	
Weight			0.66
≤3 kg	21	6	
>3 kg	42	21	
Pulmonary atresia			0.09
Yes	40	19	
No	23	8	
Diagnosis group			0.55
1	15	9	
2	11	4	
3	13	6	
4	24	8	
Size of pulmonary artery			0.49
≤5 mm	33	16	
>5 mm	30	11	
Size of graft			0.009
4 mm	43	20	
5 mm	20	7	

Our experience confirms that the MBTS provides satisfactory early palliation: the early occlusion rate is low, excessive pulmonary blood flow is extremely rare, complications are infrequent (serous leakage and seroma formation have been reported but are unusual), and take-down of the shunt at the time of complete repair is easy, particularly when a right-sided (in situs solitus) MBTS has been used.

A high early mortality rate (21%) was noted. This may be explained by a high incidence of neonates with severe congenital anomalies. Among the 13 early deaths, 11 occurred in infants less than 1 month of age, and eight deaths occurred in patients with pulmonary atresia and intact ventricular septum or with univentricular heart. Only three deaths were shunt related. Very young age and severity of the cardiac malformation have been clearly determined as major risk factors for early deaths in large series of shunting procedures [3, 7–10].

As suggested by Bove and associates [7], we used strict

Table 4. Risk Factors of Shunt Failure (Univariate and Multivariate Analyses)

Risk Factor	Univariate Analysis (log-Rank Test) *p* Value	Multivariate Analysis (Stepwise Cox Regression) *p* Value
Age	0.88	0.14
Weight	0.66	0.37
Pulmonary atresia	0.09	0.11
Diagnosis group	0.55	0.20
Size of pulmonary artery	0.49	0.60
Size of graft	0.009	0.012

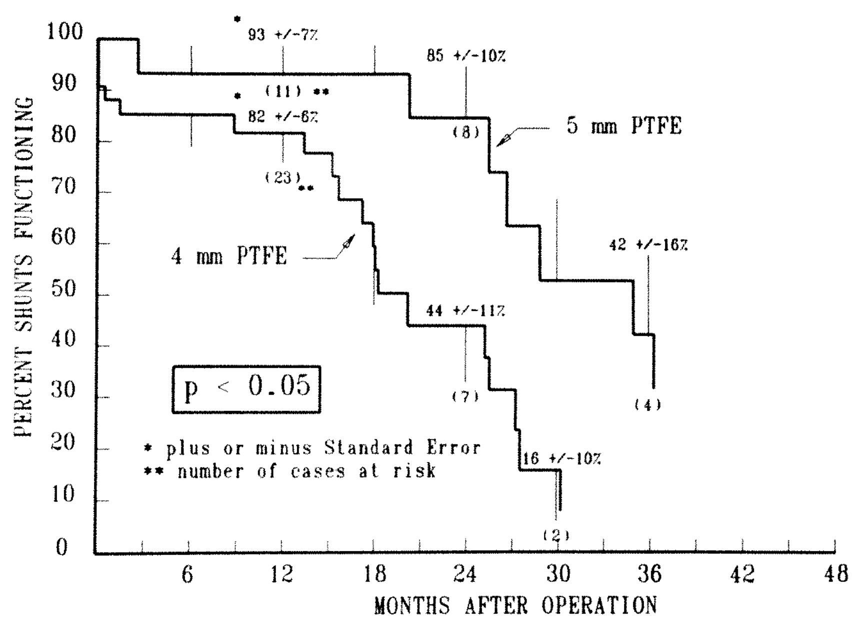

Fig 2. Actuarial probability of adequate shunt function according to the size of polytetrafluoroethylene (PTFE) graft.

criteria to assess the adequacy of late palliation. Only 58% of all shunts were functioning adequately 2 years after operation. The probability of adequate palliation was significantly higher (85% at 2 years) when a 5-mm polytetrafluoroethylene conduit was used. The relatively disappointing palliation afforded by 4-mm shunts have been reported and justified the routine use of 5-mm conduits. However, it is sometimes technically impossible to use a 5-mm graft, especially in tiny neonates with very small systemic and pulmonary vessels.

There is disagreement in the literature on the incidence of pulmonary artery distortion; rates ranging from 0% to 36% have been reported [7, 8]. In the present series, the exact incidence of pulmonary arterial distortion is difficult to determine in the absence of systematic postoperative angiographic studies. However, this incidence is probably relatively high. Among the shunts that failed (and therefore were studied angiographically) 50% showed a distortion of the pulmonary arterial branch. The stenosis may occur at the anastomosis site itself and be related to a discrepancy between the size of the pulmonary arterial branch and that of the rigid prosthetic conduit. The stenosis may also include the area before or after the anastomotic site (Fig 3) and be caused by clamp injury or failure to grow because of fibrosis of the surrounding tissue. Nevertheless, the occurrence of a severe stenosis is a shunt-related complication that may markedly increase the risk of the subsequent complete repair.

Table 5. Distortion of the Pulmonary Arterial Branch

Intracardiac Anomaly	Age at Shunt (days)	Size of Pulmonary Arterial Branch (mm)	Time of Shunt Failure (mo)	Graft Size (mm)
Tricuspid atresia	2	4	27	4
Tricuspid atresia	54	3	27	4
Tricuspid atresia	2	6	20	5
PA with IVS	20	4	15	5
PA with IVS	5	6	35	5
PA with IVS	7	4	9	4
PA with IVS	3	4	15	4
PA with VSD	22	7	20	4
PA with VSD	9	5	43	4
UVH with PA	5	5	25	5
TGA with PS	50	5	13	4
TGA with PS	15	6	19	5

IVS = intact ventricular septum; PA = pulmonary atresia; PS = pulmonary stenosis; PTFE = polytetrafluoroethylene; TGA = transposition of the great arteries; UVH = univentricular heart; VSD = ventricular septal defect.

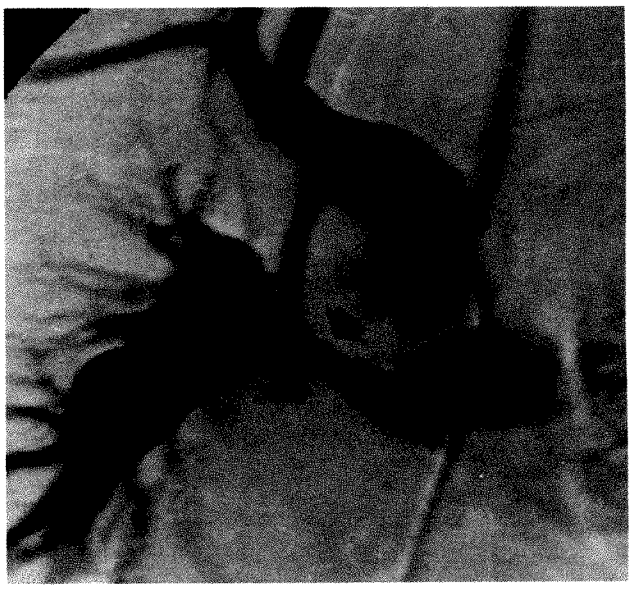

Fig 3. Severe stenosis of the pulmonary arterial branch after a right-sided modified Blalock-Taussig shunt (see text).

In conclusion, the optimal shunting procedure in infants remains to be determined. Improvements in results could be sought at least in two ways. (1) Polytetrafluoroethylene is probably a less-than-optimal material. The low rate of late adequate palliation may be due to excessive pseudointimal formation, especially with 4-mm conduits [4, 6]. The high incidence of pulmonary arterial distortion may be related at least in part to the use of a thick and rigid graft anastomosed to a small, thin vessel. To reduce these drawbacks, we are using a new biological vascular graft. The early results are encouraging, but the late results remain to be evaluated [11]. (2) An individualized approach should be used according to the type of the cardiac malformation, the anatomy of the systemic and pulmonary vessels, and the planned surgical management.

Recent reports have shown that both classic Blalock-Taussig shunts [10, 12] and central shunts [13, 14] may provide satisfactory palliation. When a long-term palliation is needed, a classic Blalock-Taussig shunt (if the size and the length of the subclavian artery are suitable) or a 5-mm MBTS should be used. If complete repair of the malformation can be planned for the patient's first year of life, a small-diameter MBTS provides adequate results. The preservation of the pulmonary artery anatomy may be of critical importance (major hypoplasia of the pulmonary arteries, planned Fontan-type repair); a central shunt is then probably a good alternative.

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Use of Aortic Valve Allografts From Hepatitis B Surface Antigen–Positive Donors

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Over a 28-month period, 31 patients undergoing aortic valve replacement received valve allografts from hepatitis B surface antigen–positive, hepatitis B e antigen–negative donors. At the time of operation, 22 recipients were immune to hepatitis B infection; 19 had antibodies to hepatitis B surface antigen, and 3 were hepatitis B surface antigen positive. Nine patients were regarded as being susceptible to hepatitis B; 7 lacked hepatitis B markers, and records of serological status could not be found for the other 2. Four of the susceptible patients received hepatitis B immunoglobulin postoperatively, and 1 of them also received one 10-μg dose of hepatitis B vaccine. The 9 susceptible patients underwent hepatitis B serological studies a mean of 17 months (range, 4 to 31 months) postoperatively. In only 1 patient, tested 29 months after operation, had antibodies to hepatitis B surface, hepatitis B core, and hepatitis B e antigens developed. He had not experienced a clinical episode of hepatitis, and results of liver function tests were normal. He had not received prophylaxis. Because hepatitis B virus may be transmitted by hepatitis B surface antigen–positive, hepatitis B e antigen–negative valves, prophylaxis should be given whenever they are used and follow-up serological studies undertaken.

(Ann Thorac Surg 1990;49:802–5)

Valve allografts have been used for aortic valve replacement at Green Lane Hospital since 1962 [1] and are accepted as a superior device with a low rate of reoperation and minimal morbidity [2–4]. Since the advent of serological testing for hepatitis B virus (HBV), donors have been screened for hepatitis B surface antigen (HB_sAg) and hepatitis B e antigen (HB_eAg). Because HB_sAg-positive material is usually not considered for implantation, we report here the use of valves from HB_sAg-positive, HB_eAg-negative donors in patients susceptible to HBV.

Material and Methods

The HBV serological findings in donors and recipients were recorded in a valve log book in the microbiology laboratory, and recipients of valves from HB_sAg-positive, HB_eAg-negative donors were identified from these records. Recipients were deemed immune to HBV if they were either HB_sAg positive or had antibody to HB_sAg at the time of operation. Patients were deemed susceptible to HBV if they lacked any serological markers for HBV or if no record of their serological status could be found. Follow-up serum from susceptible patients was obtained by each patient's general practitioner, who was informed of the reason for the request and asked if the patient had experienced any clinical or biochemical episode of hepatitis since the valve operation.

The hospital record of each patient was inspected to determine the indications for the use of the valve allograft and whether the patient had received either hepatitis B immunoglobulin (HB Ig) or hepatitis B vaccine. The presence of HB_sAg, antibody to HB_sAg, HB_eAg, antibody to HB_eAg, and antibody to hepatitis B core antigen was detected using conventional Abbott and Behring testing kits. Liver function tests were also performed on all follow-up sera.

The HB_sAg status of the blood used to prime the bypass machine from the patient who had seroconverted to HBV at follow-up was retested using the Ausab radioimmunoassay diagnostic kit (Abbott Laboratories) after the follow-up results were known.

The methods for processing heart valve allografts have been published elsewhere [5]. Donor hearts were removed as cleanly as possible at postmortem examination and then taken to a separate site where the valve was removed by aseptic technique. Valves were disinfected by immersion for 48 hours in an antibiotic medium containing cefoxitin sodium, lincomycin hydrochloride monohydrate, polymyxin B sulfate, vancomycin, and amphotericin B at 4°C. Valves were stored and sent to the theater in the gas phase of liquid nitrogen. Once thawed, a valve was discarded if not used.

Results

From June 1986 to October 1988, 31 patients undergoing aortic valve replacement received allografts from HB_sAg-positive, HB_eAg-negative donors. Twenty-two recipients (71%) were immune to HBV; 19 had antibody to HB_sAg, and 3 were HB_sAg positive. Seven patients who lacked HBV markers were clearly susceptible to HBV, and 2, for whom there was no record of HBV serological status, were assumed to be susceptible.

For the 9 patients susceptible to HBV, an allograft was considered by the surgeon to be strongly indicated on

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Table 1. Data on Patients Susceptible to Hepatitis B Infection Who Received Allograft Aortic Valves From Hepatitis B Surface Antigen–Positive, Hepatitis B e Antigen–Negative Donors

Patient No.	Sex	Age (yr)	Interval Between Operation and Follow-up Serology (mo)	Prophylaxis[a]		Preop HB_sAg or Antibody to HB_sAg	Hepatitis B Markers Postop				Liver Function Tests
				HB Ig	Vaccine		HB_sAg	Antibody to HB_sAg	Antibody to HB_cAg	Antibody to HB_eAg	
1	M	67	25	Yes	No	Nil	No	No	No	No	Normal
2	F	28	31	Yes	No	Nil	No	No	No	No	Normal
3	M	60	29	No	No	Nil	No	Yes	Yes	Yes	Normal
4	M	67	24	No	No	Nil	No	No	No	No	Normal
5	M	66	23	No	No	Nil	No	No	No	No	Normal
6	M	13	7	Yes	No	No record	No	No	No	Not done	Elevated ALP[b]
7	F	53	5	No	No	No record	No	No	No	No	Normal
8	M	61	4	Yes	Yes	Nil	No	No	No	No	Elevated ALP[b]
9	M	79	6	No	No	Nil[c]	No	No	No	No	Normal
Mean	. . .	55	17	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .

[a] Prophylaxis consisted of 400 IU of hepatitis B immunoglobulin (Commonwealth Serum Laboratories, Australia) or a 10-μg dose of H-B VAX II (Merck Sharp & Dohme). [b] The alkaline phosphatase (ALP) level was elevated, 405 U/L in patient 6 and 125 U/L in patient 8. The normal range is 25 to 100 U/L. [c] This patient was HB_sAg negative; the test for antibody to HB_sAg was not performed.

HB Ig = hepatitis B immunoglobulin; HB_cAg = hepatitis B core antigen; HB_eAg = hepatitis B e antigen; HB_sAg = hepatitis B surface antigen.

clinical grounds. The characteristics of these patients, the time between operation and follow-up, the use of prophylaxis, the preoperative and postoperative HBV serological status, and the postoperative liver function test results are shown in Table 1. The mean age of these patients was 55 years, and the mean time between operation and HBV follow-up serological study was 17 months. Four patients received prophylaxis: 3, HB Ig only and 1, HB Ig plus one dose of vaccine.

One patient had seroconverted to HBV between the operation and follow-up 29 months later. He had had no clinical episode of hepatitis, had not become a carrier, and had normal liver function. This patient had not received HBV prophylaxis. The blood used to prime the bypass machine was negative for HB_sAg on initial and subsequent testing. The surgeon's serological status was unknown.

Comment

Blood products are the main source of HBV during operation [6], but the use of tissue from HB_sAg-positive donors is another possible source of infection. There are rare reports of transmission from the surgeon [7].

For patients for whom a valve allograft is considered the most suitable replacement device, the use of material from HB_sAg-positive but HB_eAg-negative donors must be balanced against the likelihood and consequences of transmission of HBV.

It is well established that the infectivity of HB_sAg-positive serum can be correlated with the presence of HB_eAg [6, 8–11]. The presence of HB_eAg is tightly associated with the presence of HBV deoxyribonucleic acid and Dane particles in sera [6, 12–14]. The relationship between HB_eAg positivity and higher infectivity has been shown after needle-stick injuries and in mother to infant HBV transmission [6, 8–11]. Although transmission is less frequent from patients or material that are HB_eAg negative, it should be noted that the absence of HB_eAg does not rule out the possibility of transmission [15–17].

Little information is available on the use of HB_sAg-positive tissue. Three reports [18–20] comment on the transplantation of 11 kidneys from HB_sAg-positive donors. Six of 7 susceptible patients showed serological evidence of infection, including 3 who became chronic carriers. These 3 patients had received kidneys from HB_eAg-positive donors and no prophylaxis. No infected patient had clinical viral hepatitis type B and only 1 had abnormal liver function test results. It is not known whether the transplanted renal tissue itself or the blood contained in it transmitted HBV to these patients.

Transmission rates of HBV after needle-stick injuries or during birth from carrier mothers are also known. However, it is difficult to relate data from needle-stick injuries because of the uncertain nature of the extent of exposure, which ranges from nil to substantial. More reliable data are those after birth of an infant to an HB_sAg-positive mother [6, 8, 9, 11, 15, 21]. In this situation, it is known that almost every infant will be exposed to HB_sAg-positive blood and secretions [6, 11, 21], and in this setting, HB_eAg-positive mothers transmit infection to about 90% of infants, most of whom become carriers. However, for infants born to carrier mothers lacking HB_eAg, about 20% have evidence of infection at 1 year and only 6% are HB_sAg positive [11]. If the perinatal situation is used as a model for what might occur after exposure to HB_sAg-positive valves, then HB_eAg-negative material poses a significantly lower risk of transmission of HBV than HB_eAg-positive material [11].

The next factor to consider is the likelihood of illness and complications should transmission of HBV occur. In adults, most HBV infection is asymptomatic with 10% to

20% experiencing a clinical episode of hepatitis; 1% of these patients may have fulminant disease, and 10% may become chronic carriers. However, the proportion sustaining clinical illness and the proportion becoming chronic carriers may be lower among those exposed to an HB_eAg-negative inoculum [6, 11].

The rates of transmission and complication already discussed refer to situations in which no intervention was given. However, transmission rates and expression of infection can be altered by the use of prophylaxis [11, 21]. The case of mother to infant transmission serves as a model for the use of HB_sAg-positive heart valves. In the worst-case situation involving HB_eAg-positive mothers, transmission can be reduced from around 90% to 20% to 25% by the use of either vaccine or HB Ig alone and to less than 10% with both agents combined [6, 11, 21]. In infants born to carrier mothers lacking HB_eAg who receive HB Ig, the transmission rate is reduced from 20% to 4% [11]. This rate is similar to the 8.5% rate observed in infants born to HB_sAg-negative mothers in the same geographical area and probably reflects HBV infection acquired outside the neonatal period [11]. Therefore, if the neonatal situation is used as a model for what might occur with HB_sAg-positive, HB_eAg-negative valves, the use of prophylaxis, HB Ig, and vaccination should reduce the likelihood of transmission of HBV to almost zero.

The foregoing considerations led us to postulate that seroconversion and development of a carrier state would be low when HB_sAg-positive, HB_eAg-negative valve allografts were transplanted into recipients susceptible to HBV and that clinical infection would probably not occur. In this series, 1 of 9 patients seroconverted to HBV, and this patient was 1 of the 5 who did not receive prophylaxis. Although this series is small, it encourages us to continue to use such material but to insist that appropriate prophylaxis be given. This should consist of HB Ig and vaccine at the time of operation followed by completion of the vaccine schedule.

These observations are important because of the scarcity of allograft valve donors, the relatively high prevalence of HBV infection in New Zealand and some other countries, and the need, if this policy is not followed, to discard these otherwise suitable donor valves. During the period of this study, 8% of harvested valves were from HB_sAg-positive donors. Hepatitis B surface antigen–positive sera should also be tested for delta (hepatitis D virus) markers; if they are present, the valve should be discarded. This would prevent coinfection of a seronegative recipient or superinfection in a carrier and would remove the possibility of severe acute or chronic liver disease due to both viruses [6].

If a patient is likely to be best served by a valve allograft, then consideration can be given to vaccination before operation. New Zealand has begun a national HBV immunization program so that in the future, more recipients should be immune to hepatitis B. However, it may still be prudent to use a booster dose of vaccine or HB Ig before the use of HB_sAg-positive, HB_eAg-negative valves.

The information in this study is incomplete in two relatively minor respects. The serological status of the surgeon involved was not known, and for 2 recipients, the interval between operation and follow-up serological study was just inside the accepted maximum incubation period for HBV of 160 days.

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REVIEW OF RECENT BOOKS

Heart and Heart-Lung Transplantation

Edited by John Wallwork
Philadelphia, W.B. Saunders, 1989
566 pp, illustrated, $99.00

Reviewed by Leslie W. Miller, MD

The book *Heart and Heart-Lung Transplantation* by Wallwork is a good addition to the field of cardiac transplantation with several outstanding chapters. The book is fairly complete, but in being so, has perhaps included several chapters that do not add much to the overall value of the book, such as chapters on procurement, anesthesia, pathology of the excised heart, and donor transplanted disease. In addition, several chapters seem to be out of sequence such as the chapter on registry data, the discussion of rejection by Dr Morris, and the chapter on pathology of the excised lung in the middle of the section on heart transplantation. There seemed to be some chapters that were also too long, such as "Quality of Life Indicators," which seemed excessive at 33 pages in comparison with the chapter on transplant coronary artery disease, the number one cause of death after heart transplantation, which was only 7 pages.

The chapter on immunosuppression was somewhat disappointing as immunosuppression is one of the major aspects in the field of heart transplantation today. This chapter gave a relatively brief outline and history of immunosuppressive regimens employed, but did not discuss in any detail the comparative benefits attributed to use of induction versus noninduction therapy or give a substantial review of the side effects of agents now available. The chapter by Baumgartner, which included practical clinical management of cyclosporine, in some way overlapped this deficit, but it too could have used more extensive review of this very critical agent.

One chapter that was somewhat unique to this book was the discussion of the implications of resource allocation with heart and heart-lung transplantation. The authors do a thorough job of reviewing some detailed numbers of the costs of this procedure within the United Kingdom and discuss the relative pros and cons of the current allocation of this procedure. This topic will perhaps become even more critical in the budgetary constraints ahead in the United States as well as Europe and was a very pleasant surprise and relatively important contribution to this book.

The section on heart-lung transplantation is very good, and in general, somewhat superior to the section on heart transplantation. All the chapters are complete and provide a good overview, with the possible exception of the chapter on the surgical procedure itself. Although the author obviously provides a good technical description of the operation, it was brief and did not include comment on the potential complications, the merits of en bloc versus staged resection, modes and relative merits of the various forms of preservation, or the relative problems encountered with previous thoracic operations. The sections on the diagnosis of rejection as well as the use of biopsy for diagnosing rejection were excellent, as was the overview of the physiology of the transplanted lung, which seemed to have been more appropriate at the beginning of the section rather than at the end.

In general, this book offers a fairly good overview of the field of heart and heart-lung transplantation today. In particular, it is perhaps the best reference on the topic of heart-lung transplantation currently available. The book is well referenced and describes nearly every aspect of transplantation.

St. Louis, Missouri

Use of Gianturco Self-Expandable Stents in the Tracheobronchial Tree

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Gianturco self-expandable stents were used successfully in the management of 5 patients with tracheobronchial pathology. Placement was performed under endoscopic and fluoroscopic guidance. None of the patients has experienced complications secondary to the stent placement, and in all of them the clinical problems resolved satisfactorily. Longer follow-up is required to determine the place of tracheobronchial stenting in patients with respiratory compromise.

(Ann Thorac Surg 1990;49:806–9)

Use of the Gianturco self-expandable stent was initially intended for treatment of stenosis within the vascular system [1–5]. Subsequently, the stent has been used for treatment of biliary strictures [6]. Several groups of investigators have also described use of this stent in tracheomalacia that has occurred after tracheal resection [7–9]. We describe the use of the Gianturco stent for treatment of tracheobronchial tree pathology in 5 patients.

Material and Methods

The Gianturco stent (Cook Inc, Bloomington, IN) is made of 0.07-mm diameter stainless-steel wire laced into a zigzag cylinder that spontaneously expands on delivery. Different diameter stents are available; commonly, two Gianturco stents are joined together with lateral barbs affixed to the stent to minimize migration in the tracheobronchial tree (Fig 1). For stent placement, the patients received general anesthesia and the tracheal lesions were first examined with a rigid bronchoscope passed through the vocal cords. The exact position of the tracheal stenosis was established using a combination of endoscopic and fluoroscopic guidance, defining the distal and proximal limits of the lesion. If severe inflammatory changes were observed at the level of the stenosis, the distal end of the stent was placed clearly beyond the site of obstruction to optimize stent position. Thus, the stents placed were always longer than the lesion. Two or more stents may be needed in some patients. Metallic markers are placed on the skin, specifying the level of the lesion, and the remainder of the procedure is performed with fluoroscopic guidance alone. A 12F Teflon sheath for introduction of the stent is then passed through the bronchoscope, placing its tip beyond the most distal aspect of the stricture.

Under fluoroscopic control, the stent is advanced through the Teflon sheath until it reaches the distal end of the sheath. The pusher is held with one hand, keeping the stent at the desired level, and the Teflon sheath is slowly removed, allowing the distal aspect of the stent to expand. Once the first stent is totally extruded from the Teflon sheath, the level of the stent relative to the metallic skin markers is checked, verifying the level of the stricture. If the stent is not at the desired level, its position is minimally adjusted by gently advancing the stent forward or pulling it back into the sheath. Further retraction of the Teflon sheath releases the second stent. Once the second stent is in place, the Teflon sheath is removed and the optical lens of the bronchoscope is introduced to assess the anatomical relationship of the stent to the tracheal stenosis. Baseline chest roentgenograms are then taken for comparison with subsequent studies.

Case Reports

Patient 1

A 71-year-old man with severe cardiovascular disease and epidermoid carcinoma of the vocal cords underwent total laryngectomy and postoperative radiation therapy to the neck and upper mediastinum. Twelve months after operation, the patient had dyspnea on exertion and stridor. Fiberoptic bronchoscopy showed narrowing with severe inflammation in the lateral and posterior wall of the trachea at the level of the tip of the tracheostomy cannula. A biopsy showed no malignancy. The patient's condition deteriorated and he required mechanical ventilation through a No. 5 pediatric endotracheal tube.

Under general anesthesia, a rigid bronchoscope was passed through the tracheostomy. A 20-mm Gianturco stent was placed at the site of tracheal stenosis. In the period immediately after the procedure, the tracheal lumen was widely patent, measuring 70% to 80% of the

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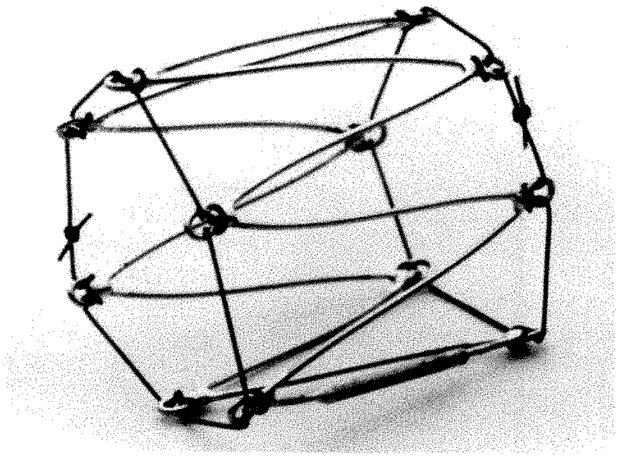

Fig 1. Gianturco barbed stent.

original tracheal diameter. Accurate positioning of the stent was directly confirmed with flexible bronchoscopy and with chest radiography (Figs 2, 3). The patient was discharged 48 hours after stent placement with a tracheostomy tube positioned with its tip bridging the stent. One month after the procedure, the patient had no respiratory symptoms; at that time, endoscopy revealed epithelization of the stent wires. The chest roentgenogram showed clearing of left lung infiltrates and further expansion of the stents (Fig 4). The tracheostomy was removed. The patient has remained asymptomatic for 12 months.

Patient 2

A 58-year-old man with posttraumatic hydrocephalus requiring ventriculoperitoneal decompression was seen with tracheal stenosis as a complication of tracheostomy performed 2 years earlier. The luminal narrowing of the trachea was 70%. A 30-mm Gianturco stent was placed through the sheath of a rigid bronchoscope passed through the vocal cords; it was placed across the stenosis under fluoroscopic control, and the tracheostomy was closed. The patient has remained without respiratory symptoms for 12 months. Radiological and endoscopic studies at 3 months have shown no evidence of recurrence of the stenosis and epithelization of the stent wires.

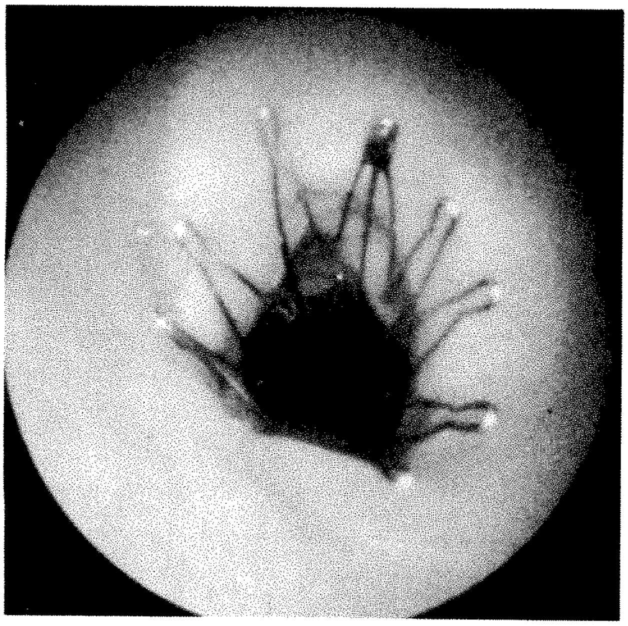

Fig 2. Endoscopic view of Gianturco stent at the level of the tracheal stenoses.

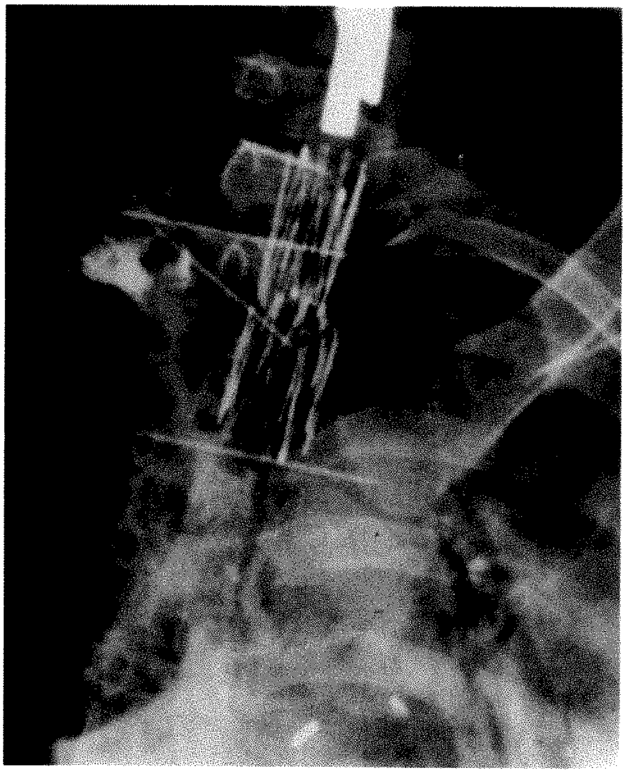

Fig 3. Fluoroscopic image showing the stent in place with endoscope viewing its proximal end. Expansive consolidations of the left lung are evident. Opaque markers were placed before stent introduction for guidance purposes under fluoroscopy.

Patient 3

A 60-year-old woman with an epidermoid carcinoma of the midesophagus underwent partial esophagectomy, after which a tracheoesophageal fistula developed. A cervical esophagostomy was performed for control of the fistula; this was complicated by periincisional abscess. Necrosis of the trachea at the level of the fistula also occurred, requiring placement of an indwelling tracheostomy cannula. Subsequent to these infectious processes, the tracheoesophageal fistula reappeared and a permanent gastrostomy was performed to isolate the fistulous tract; the tracheostomy cannula was exchanged for a Montgomery T tube. Six months later, gastrointestinal continuity was reestablished with a colonic interposition. After an additional 6 months, the Montgomery T tube was replaced with a 30-mm double Gianturco stent. The patient required mechanical ventilation for five days but has had no respiratory symptoms for the subsequent 9 months.

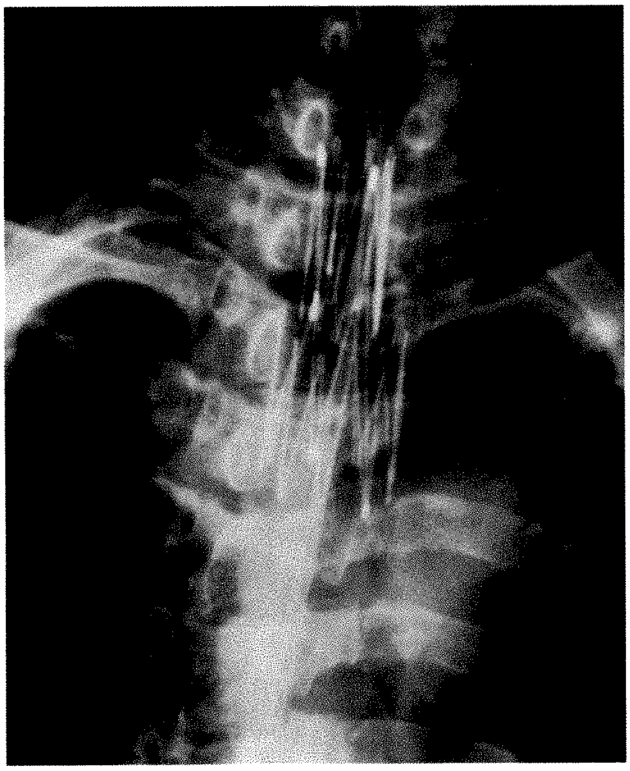

Fig 4. Follow-up chest roentgenogram shows clearing of left lung infiltration and further expansion of the stent.

Patient 4

A 60-year-old man had tracheal stenosis secondary to long-term tracheostomy placement necessitated by respiratory complications after a vascular and abdominal operation. A 30-mm diameter double-barbed stent was placed using a combined bronchoscopic and fluoroscopic approach. The tracheostomy was closed seven days after stent insertion. At 6-month follow-up, the patient has no respiratory problems.

Patient 5

An 86-year-old man had high tracheal stenosis after tracheostomy. A 20-mm diameter double-barbed stent was placed. The placement of this first stent was distal to the stricture. A second 20-mm stent was then placed more proximal. No symptoms related to the position of the stent in the vicinity of the vocal cords have occurred. The respiratory symptoms disappeared at 2-month follow-up.

Comment

The current method of treating patients with tracheal or bronchial stenoses is surgical, with resection of the affected area and end-to-end anastomosis. Attempts are made to perform the anastomosis without tension to prevent complications [10, 11]. Operative techniques are limited when resection of longer segments of trachea and/or bronchus is needed, and operation frequently results in tracheomalacia of the interposed graft and respiratory compromise. Alternative treatment of longer stenoses depends on transendoscopic laser resection of tumors [12] and placement of silicone endoprostheses [13] or Montgomery T tubes [14]. Placement of metallic stents by a combination of endoscopic and fluoroscopic guidance is another alternative treatment for tracheobronchial stenosis [7, 8].

Metallic stents have been used extensively in biliary ducts [15] and in veins [16] without evidence of erosion in long-term follow-up in either laboratory animals or patients. Endothelization occurs in the vascular tree [16]. Infection has not been a problem when the stent is placed under sterile conditions [15, 16].

The main disadvantage of these stents is that they cannot be removed endoscopically. They can, however, be removed at the time of surgical exploration if surgical reconstruction is attempted. A removable type of stent is currently under investigation in laboratory animals.

Placement of these stents requires attention to technical details. The level of the stenosis must be precisely defined. If the trachea does not follow a straight path, placement of the Teflon sheath can be facilitated by passing it over a 0.89 mm (0.035-in.) J guidewire advanced beyond the lesion. Initial release of the distal end of the stent can be quite abrupt, tending to place it beyond the desired level in the tracheobronchial tree. Attention to detail during release is of utmost importance, because the position of the stent cannot be adjusted once the stent has been released. If placement is inadequate, a second stent must be placed. Migration of the stent is prevented by the barbs. This appears to be particularly true in patients with severe stenosis such as our patient 3.

The use of metallic self-expandable stents in tracheobronchial pathology is indicated in patients who have medical contraindications to a surgical procedure. Candidates include patients with diffuse or focal inflammatory disease, primary or secondary malignancies, extrinsic compression of the tracheobronchial tree, primary or secondary tracheomalacia, and postreconstructive airway collapse or narrowing. Stent placement can also be used as an alternative or complement to other therapeutic modalities, such as laser resection, electrocoagulation, and cryocoagulation, and in conjunction with placement of other tracheal devices, such as the Montgomery T tube. The ultimate role of this particular interventional procedure in tracheobronchial pathology will likely be better defined with time.

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INVITED COMMENTARY

Surgical resection and primary reconstruction remains the goal of airway surgery whenever possible. Until the ideal tracheal prosthesis is found or tracheal transplants become feasible, a cautious, open mind must be maintained about "other possibilities." Tracheostomy tubes and Montgomery T tubes are suitable for most patients when surgical resection is not feasible. Their greatest advantage is ease of care and removability, thereby not precluding later surgical resection and reconstruction. The stent described by Varela and associates may be useful in certain highly selected instances when surgical resection is not contemplated. Its major drawbacks would seem to be lack of long-term follow-up and its inability to be removed as currently designed. Caution must also be exercised in recommending it for "focal inflammatory disease [and] primary or secondary malignancies" as Varela and associates claim. If more experience and longer follow-up find it safe, it may represent another alternative for the airway surgeon to have at his or her disposal.

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Management of Extensive Right Ventricular Injury or Rupture

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Most penetrating right ventricular injuries require simple suture repair, but more extensive injury or rupture of the right ventricle may not be amenable to this method. We have developed an approach to the problem and a technique for repair. Compression of the area with early institution of cardiopulmonary bypass will result in decompression of the right ventricle and preservation of perfusion, preventing profound hypotension. Coverage with an onlay autologous tissue patch provides hemostatic control of the defect without compromising ventricular function. Reinforcement with omentum or muscle flap can give additional protection when risk of infection is present. Application of these principles can be lifesaving and insure good cardiac function despite massive injury to the right ventricular myocardium.

(Ann Thorac Surg 1990;49:810–3)

Although small penetrating wounds involving the ventricular myocardium can be repaired routinely by placement of sutures beneath the surgeon's finger [1–5], larger tears or rupture of the right ventricle may be difficult to manage and cause fatal injury [6, 7]. Such injuries may not be amenable to suture repair, particularly if the adjacent ventricular tissue is compromised by contusion, local infection, or other conditions. We have had successful experience with 2 patients in whom rupture of the right ventricle was repaired by placement of a tissue patch, and management of these patients has allowed us to develop an approach to this problem.

Material and Methods

Two patients were recently treated by our service on an emergency basis for tears to the ventricular myocardium. A 73-year-old man was admitted to the hospital with unstable angina pectoris. He had a history of steroid-dependent chronic obstructive pulmonary disease. Cardiac catheterization showed an 80% stenosis of the left anterior descending coronary artery with near-total occlusion of the right coronary artery and mild irregularities of the circumflex. During balloon angioplasty, complete occlusion of the left anterior descending coronary artery and cardiac arrest with ventricular fibrillation developed. The patient was intubated, and closed-chest resuscitation was initiated. Defibrillation attempts were unsuccessful, and he was transferred to the operating room with chest compression in progress.

An immediate sternotomy incision was made, and open cardiac massage was performed while cannulas for cardiopulmonary bypass were introduced into the aorta and right atrium. During the massage, the right ventricular outflow tract was perforated by the assistant's thumb (Fig 1). Bypass was instituted, and a single coronary bypass graft was constructed to the left anterior descending coronary artery. The right ventricle was repaired with Ethibon (Ethicon, Inc, Somerville, NJ) felt-pledgetted sutures, but when the heart was allowed to fill, the patient had persistent bleeding from the ventricular defect. A patch of anterior pericardium was obtained and secured to the myocardium around the tear using interrupted Prolene (Ethicon) sutures reinforced with Teflon felt while approximating the edges of the torn ventricular myocardium beneath the patch (Fig 2). An intraaortic balloon was inserted, and the patient was weaned from cardiopulmonary bypass.

Three-vessel coronary bypass grafting for unstable angina pectoris with placement of the left internal mammary artery to the left anterior descending coronary artery was performed in a 53-year-old man. His history included clinically significant chronic obstructive pulmonary disease and a cholecystectomy through a right subcostal incision. His initial postoperative course was uneventful, but on postoperative day 7 sternal wound dehiscence developed. Cultures and a Gram stain of the fluid were initially negative, and the patient was closed again with vertical and horizontal wires. Late cultures grew *Staphylococcus epidermidis*.

The patient's wound failed to heal, and he was returned to the operating room after six days for debridement and removal of the wires. His wound was packed open, and we anticipated closure with muscle flaps in three to five days. Two days after debridement, he vomited and massive bleeding through his dressings developed. He was hypotensive, with a systolic blood pressure of approximately 80 mm Hg, and was transferred to the operating room with compression of the sternal wound. The femoral artery and vein were exposed and cannulated after heparinization. Removal of the packs showed that the

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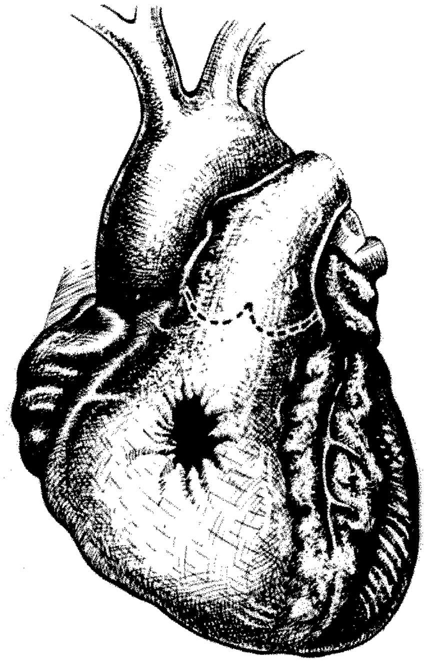

Fig 1. Perforation of right ventricular outflow tract during open cardiac massage.

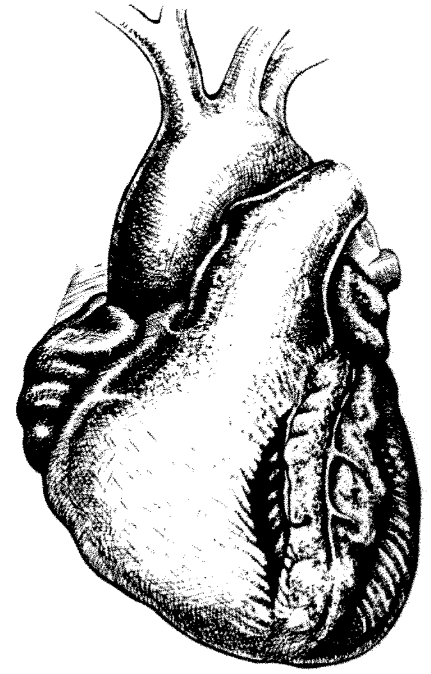

Fig 3. Right ventricular tear in patient with open sternotomy incision after debridement for sternal wound infection.

patient had a 7-cm tear in the anterior wall of the right ventricle adjacent to the interventricular septum (Fig 3).

The bleeding was controlled with cardiotomy suction cannulas, and the left ventricle was dissected free from its adhesions to the anterior chest wall. The left anterior descending coronary artery was identified, and the edges of the defect were reapproximated with sutures reinforced with Teflon felt pledgets, but the defect could not be closed completely. Because of his recent operation, no pericardium was available for buttressing the repair. The incision was extended caudad, and the left anterior rectus sheath was exposed. A 10 × 10-cm patch of fascia was

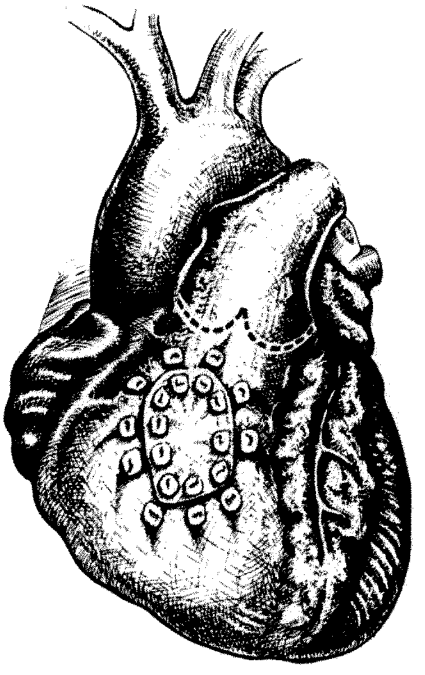

Fig 2. Repair of right ventricular perforation with onlay patch of autologous pericardium.

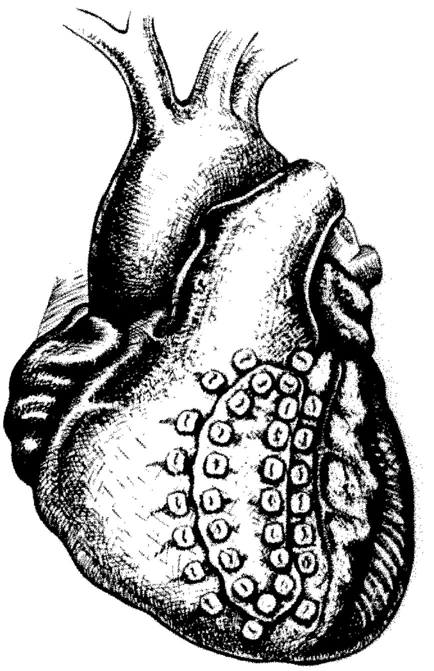

Fig 4. Repair of right ventricular tear with autologous patch from anterior rectus sheath.

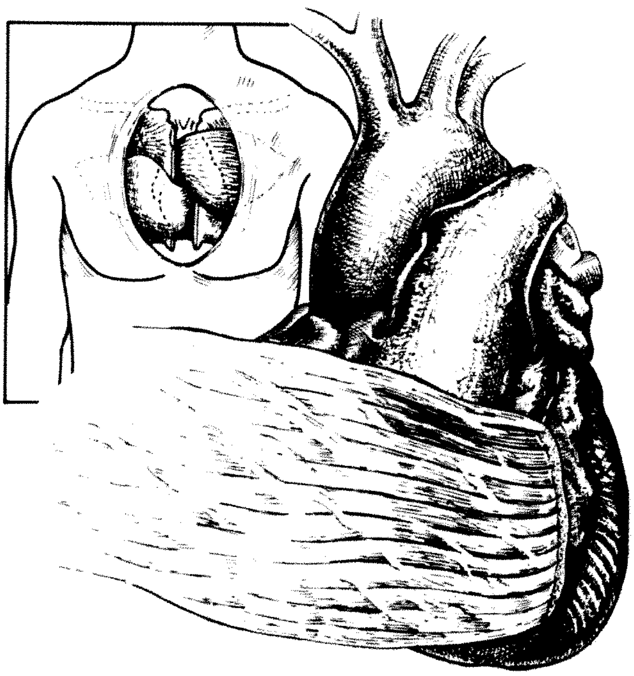

Fig 5. Placement of bilateral pectoralis major muscle flaps to facilitate sternal wound healing and protect right ventricular repair.

excised and used to cover the defect (Fig 4). The patient was weaned from cardiopulmonary bypass without inotropic support and maintained satisfactory blood pressure and cardiac output with normal pulmonary artery pressures.

Because of his previous abdominal operation and the use of the internal mammary artery, we believed that neither omentum nor rectus muscle would be available for reinforcement of the patch closure. After mobilization of both pectoralis major muscles, we placed the right pectoralis major over the right sternal edge and secured it to the epicardium to cover the patch. The left pectoralis muscle was used to cover the left sternal edge and to fill the remaining space in the mediastinum (Fig 5).

Results

The intraaortic balloon was removed from the first patient after 36 hours, and a postoperative electrocardiogram showed nonspecific ST-T wave changes but no evidence of transmural infarction. The patient required ventilatory support for six days and was discharged from the hospital on postoperative day 16. He had no neurological sequelae and remains asymptomatic of cardiac disease after 3 years. The second patient made an uneventful recovery and was discharged from the hospital on postoperative day 21 with a well-healed incision and no cardiac symptoms.

Comment

Minor injury to the ventricular myocardium can frequently be repaired with simple sutures [1–5], although more extensive injuries, particularly to the thin-walled right ventricle, may not be amenable to this technique. The right ventricular myocardium may not hold suture material well and can be particularly difficult to control unless decompression is provided by cardiopulmonary bypass. Although some investigators have advocated use of inflow occlusion to allow repair of the defects [2], refilling of the right ventricle and return of pressure may cause persistent bleeding or further injury to the adjacent muscle.

Because of the absence of major coronary arteries on the anterior surface of the right ventricle in most patients, an onlay patch can be applied without risk of coronary occlusion and infarction. The low-pressure right side of the heart can function adequately even after extensive injury to the anterior wall of the right ventricle and placement of a noncontractile patch. The pericardium is generally available to make a patch so that synthetic material, as described for repair of left ventricle defects, is avoided [8, 9]. Pericardial patches in the right ventricular outflow tract have been used in repair of congenital heart defects for some time [7]. In patients who have had previous cardiac operations, the pericardium may not be available and fascia can be obtained from the anterior rectus sheath, as described in the second patient. The risk of infection in this patient prompted us to apply additional means to protect the right ventricular repair from subsequent infection. Particularly in the presence of infection, omentum has been used frequently to repair intrathoracic defects in patients who have not had an intraabdominal operation [10–13]. It appears able to develop neovascularization in tissue to which it is applied and has particular resistance to infection, as well as the ability to limit ongoing infection [13].

In patients who have had a previous abdominal operation, the omentum may be bound by adhesions; muscle flap placement provides an excellent alternative for repair and closure of the mediastinal space in infected sternal wounds [14–16]. Blood supply to the rectus muscles in patients in whom one or both internal mammary arteries have been used for coronary revascularization will be based on the inferior epigastric artery, preventing use of rectus muscle for coverage of the heart and closure of the mediastinal space. The pectoralis major muscles can be mobilized from their attachment to the humerus and provide a large, well-vascularized, broad-based muscle flap for repair of the sternal defect, as well as coverage of the right ventricle [16].

Even though the injuries we have described represent unusual circumstances, the onlay patch technique can be used to repair right ventricular injury that may occur when the sternal incision is opened for reoperative cardiac surgery. The right ventricle can be damaged by the sternal saw [17] or excessive traction on the left sternal edge during dissection of the myocardial adhesions. When major bleeding develops in a patient with an open sternal wound after vomiting or coughing subsequent to debridement and open packing, the possibility of major right ventricular injury must be recognized. It is essential to leave the packing in place and compress the wound until cardiopulmonary bypass is available. Premature removal of the packs to assess the extent of the injury may result in uncontrollable bleeding and profound hypotension.

In patients who have a suggestion of extensive myocardial injury, especially associated with myocardial contusion, infection, steroids, or other causes, simple suture techniques may be inadequate, may increase the extent of the defect, and may limit the size of the right ventricular chamber if large gathering sutures are used. Early institution of cardiopulmonary bypass allows decompression of the right ventricle and avoids extending the injury with unsuccessful attempts at repair. The onlay patch technique described may be used as an adjunct to control bleeding.

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Unusually Early Recoarctation After Extended End-to-End Anastomosis in a Neonate

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A seven-day-old neonate with aortic coarctation and hypoplastic aortic arch underwent extended end-to-end repair with no residual gradient. Eleven hours after repair, femoral pulses were not palpable and recoarctation was confirmed by echocardiography. After failure of prostaglandin E_2 infusion, a radically extended end-to-end anastomosis was performed eight days after initial repair. No recoarctation was evident 10 months after the second repair. The unusually early recoarctation was presumably due to residual and active duct tissue in the repair margins.

(Ann Thorac Surg 1990;49:814–5)

The association between coarctation of the aorta and arch hypoplasia in the neonate can be difficult to manage surgically. Recoarctation, which develops more frequently in patients undergoing neonatal repair, remains a problem [1]. We present a case in which there was documented absence of transanastomotic gradient at the end of operation, yet 11 hours postoperatively signs of recoarctation had developed, which were later confirmed by two-dimensional echocardiography and at reoperation eight days later.

A seven-day-old male infant was transferred from another hospital where a clinical diagnosis of coarctation of the aorta had been made. On arrival at our institution, he showed signs of very low cardiac output. His right arm pulse was normal but the left arm pulse was diminished. Femoral pulses were not palpable. He had marked tachypnea and respiratory distress. The liver was palpable 4 cm below the costal margin. He was acidotic and anuric (pH, 7.22; base excess, −16). Echocardiography revealed a diagnosis of severe aortic coarctation with hypoplastic aortic arch and an associated small to moderate perimembranous ventricular septal defect. The patient was paralyzed, ventilated, and resuscitated with prostaglandin E_2, after which he was taken to the operating room for urgent repair.

At operation a preductal coarctation with local complete atresia was found. The aortic arch was moderately hypoplastic. The ductus arteriosus was ligated after full mobilization of the aorta. Vascular clamps were applied (Fig 1), the coarctation was resected, and an extended end-to-end anastomosis (method of Zannini and associates [2]) was performed with use of continuous 6-0 polypropylene suture. Immediately after the repair there was no pressure gradient between the right radial artery (95/50 mm Hg) and the descending aorta (95/55 mm Hg).

Only 11 hours after repair, the femoral pulses were noted to be weak and the arm/leg pressure gradient had increased from 0 to 30 mm Hg. Infusion of prostaglandin E_2 was resumed, and the gradient fell to less than 10 mm Hg. Infusion of prostaglandin E_2 was discontinued 40 hours later, after which the gradient remained between 5 and 20 mm Hg for two days, gradually increasing thereafter to between 20 and 30 mm Hg. The child underwent repeat echocardiography, which suggested serious recoarctation.

A decision was made to reoperate, and at reoperation (eight days after initial repair), a 60-mm Hg gradient was found across the anastomosis. After extensive mobilization, a more radical end-to-end repair [3] was performed with the help of special (Elliott) coarctation clamps (Codman Ltd, Maidenhead, England) (Fig 2). When the vessels were opened, there was some friable, possibly ductal, tissue around the proximal margins of the incision but, as it extended well across the inferior surface of the narrow arch, it could not be resected. The two ends were approximated and sutured together with use of continuous 7-0 polypropylene suture.

After repair there was a systolic pressure gradient of 20 mm Hg across the anastomosis, but a good pressure waveform was observed in the descending aorta. Postoperatively, femoral pulses were good and echocardiography confirmed a satisfactory repair. No recoarctation (as judged by a 0-mm Hg arm-leg blood pressure gradient and the absence of Doppler echocardiographic gradient) had occurred 10 months after the second operation.

Comment

We reviewed recently our experience in 109 neonates with coarctation of the aorta. Recoarctation developed in 24 patients, occurring between 1 month and 24 months after repair [1]. We have not seen another case like this patient, in whom genuine recoarctation developed as early as 11 hours after repair. Differentiation between residual (left at the time of operation) and recurrent (occurring after complete surgical relief of coarctation) coarctation can be difficult in retrospect if intraoperative (after repair) pressures are not measured. In this case no gradient was observed in the operating room between the right radial

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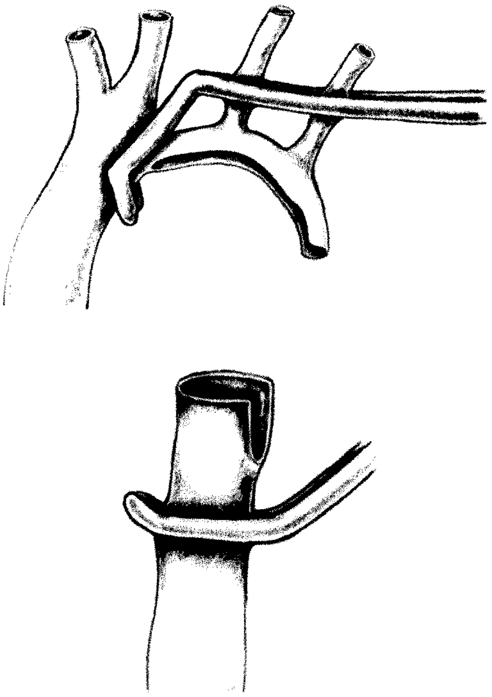

Fig 1. Method of Zannini and associates [2].

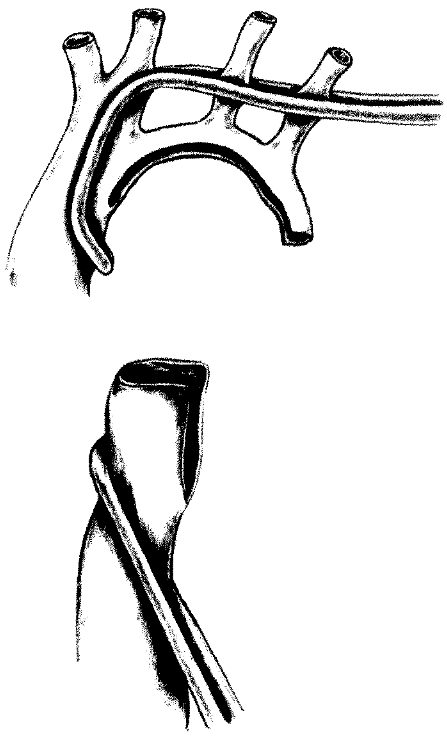

Fig 2. Radical end-to-end repair (Elliott [3]). Note the proximal position of the upper clamp and long incisions.

and descending aorta, despite the later findings, suggesting true recoarctation rather than residual coarctation.

Why then did such early recoarctation develop in this patient? Although an extended end-to-end aortic arch anastomosis was performed at initial repair, and as much ductal and abnormal tissue as possible was resected [2], it seems likely that some ductal tissue must have been left behind in the margins. This view is supported by the fact that the signs of recoarctation improved in response to the intravenous infusion of prostaglandin E_2 postoperatively. Certainly ductal tissue may be found well around the aortic arch in some specimens [4].

At the second operation there was a 60-mm Hg gradient across the anastomosis. We chose to use a modified extended end-to-end anastomotic technique [3] for the second repair, which allowed us to incise the undersurface of the aortic arch much more extensively around to the left wall of the ascending aorta and, by creating a relaxing incision in the lateral aorta, to make a wider anastomosis.

Thus, very early recoarctation after extended aortic arch repair can occur and femoral pulses must be examined frequently after operation. If signs of recoarctation do develop, administration of prostaglandin E_2 should be started on the assumption of residual ductal tissue. Further operation may be required.

However, prevention is better than cure; thus for the patient with coarctation and hypoplastic aortic arch, an initial radical repair may be the best option.

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Myocardial Revascularization With the Lateral Costal Artery

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The lateral costal artery is a branch of the internal mammary artery that occurs in 27% of cadaver series. The similarities to the internal mammary artery and its easy accessibility would suggest its use as a bypass conduit for coronary revascularization. We describe the harvesting and utilization of this artery as an in situ coronary bypass graft, and we have initiated a study examining the exact frequency of this anomalous vessel. The availability of the lateral costal artery either unilaterally or bilaterally adds another dimension to the concept of complete arterial conduit myocardial revascularization.

(Ann Thorac Surg 1990;49:816–8)

The first description of the lateral costal artery was in 1730 by Heister, who called it the lateral internal mammary artery. The famous anatomist Henle described it further as "arising from the internal mammary near its entrance into the thorax and descending on the inner surface of four to six upper ribs and anastomosing with the corresponding intercostal arteries"[1].

In 1951, Benjamin N. Kropp, an anatomist from the Queen's University Medical School in Kingston, Ontario, published the definitive report on the lateral costal artery [2]. The emphasis was on the various nomenclature and its precarious location in terms of thoracentesis and surgical procedures. Kropp reported his examination of 112 cadavers. All were white, 104 male and 8 female. The artery was identified in 31 cadavers or 27.6%, either unilaterally or bilaterally. When identified unilaterally, the left side was more common. If present on both sides of the chest, the length was not necessarily the same, and the number of interspaces traversed could vary within the same subject.

The lateral costal artery is described as usually arising from the internal thoracic artery (Fig 1) but has also been reported to originate separately from the subclavian or supreme intercostal arteries [3]. It courses the chest usually in the midaxillary line, though it can have a variable location that will bring it more anteriorly. It has been observed that if it traverses six intercostal spaces, the caliber will be as large as the internal mammary artery. There are usually two venae comitantes that accompany the artery, and these drain into the subclavian vein. At each interspace, branches are given off to the intercostal artery anteriorly and posteriorly.

In Kropp's series of cadavers, slightly less than half of the lateral costal arteries reached the fifth or sixth intercostal space. Only a third ended at the first or second intercostal space. If Kropp's observed incidence of this anomaly is correct, then optimistically, we could consider 10% to 15% of patients who are candidates for operation as likely to have a lateral costal artery that is usable for coronary bypass grafting.

We describe the utilization of this artery for myocardial revascularization in conjunction with both internal mammary arteries. Although this concept originally began with a single patient, we are now studying the incidence of this anomalous artery, advocating its use in a greater number of patients.

A patient with two-vessel coronary artery disease recently underwent bilateral internal mammary artery revascularization. During the harvesting, a large artery originating from the proximal portion of the internal mammary artery

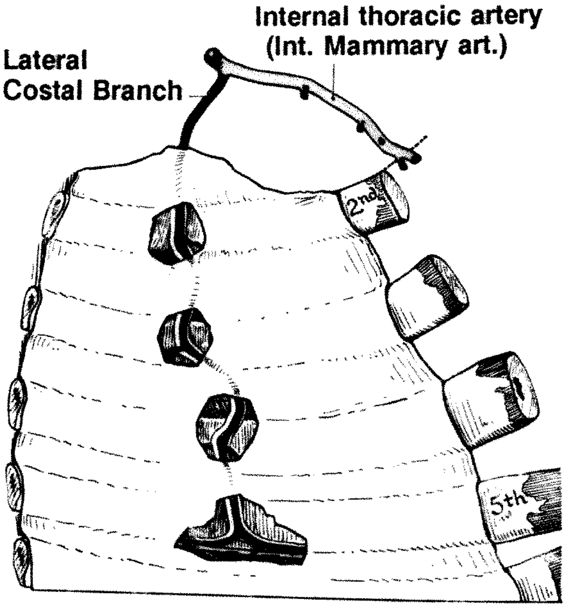

Fig 1. Lateral costal artery. (Reprinted from Grant JCB, Grant's Atlas of Anatomy, *6th edition. © 1972, The Williams & Wilkins Company. Reproduced by permission.)*

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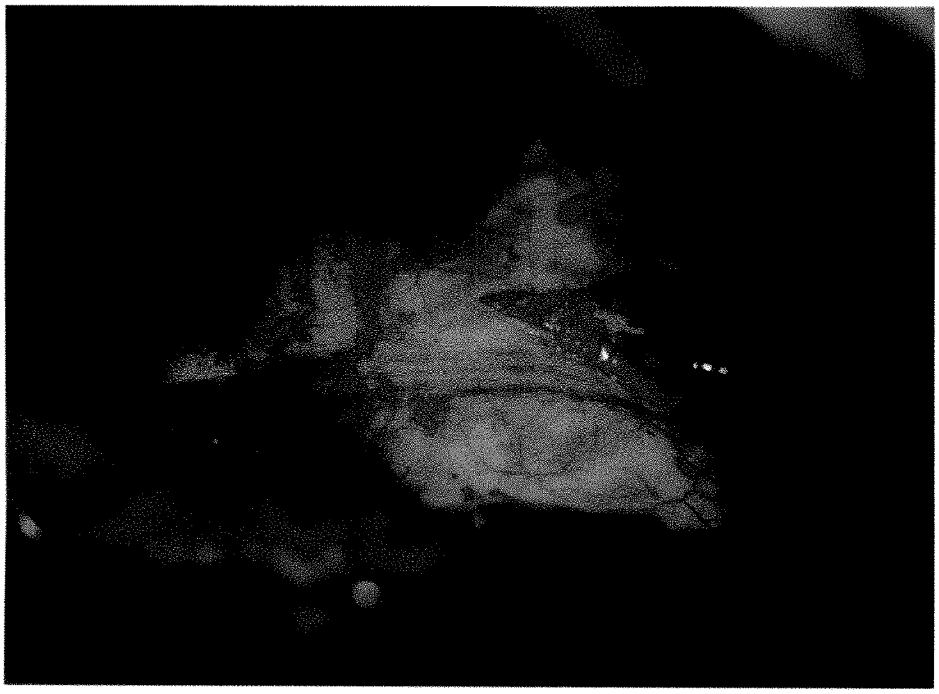

Fig 2. Right lateral costal artery with accompanying venae comitantes.

was observed. It was bilateral with the right larger than the left. It traversed the lateral aspect of each hemithorax in the midaxillary line, with two accompanying venae comitantes on either side (Fig 2). Initially, they were thought to be accessory internal mammary arteries, but they were later proved to be lateral costal arteries. This artery was harvested as a pedicle, as one would do for an internal mammary artery graft (Fig 3). The outer diameter was 2 mm and the flow after papaverine injection was 100 mL/min. The total length of the lateral costal artery was 20 cm. This artery was used to bypass the diagonal branch, constructing the anastomosis with 8-0 Prolene (Ethicon, Somerville, NJ) suture. Additional revascularization was performed by anastomosing the right internal mammary artery with a flow of 270 mL/min to the right coronary artery, and the left internal mammary artery with a flow of 300 mL/min to the left anterior descending coronary artery. There were no intraoperative difficulties and a repeat cardiac catherization was performed before discharge to demonstrate the lateral costal artery conduit

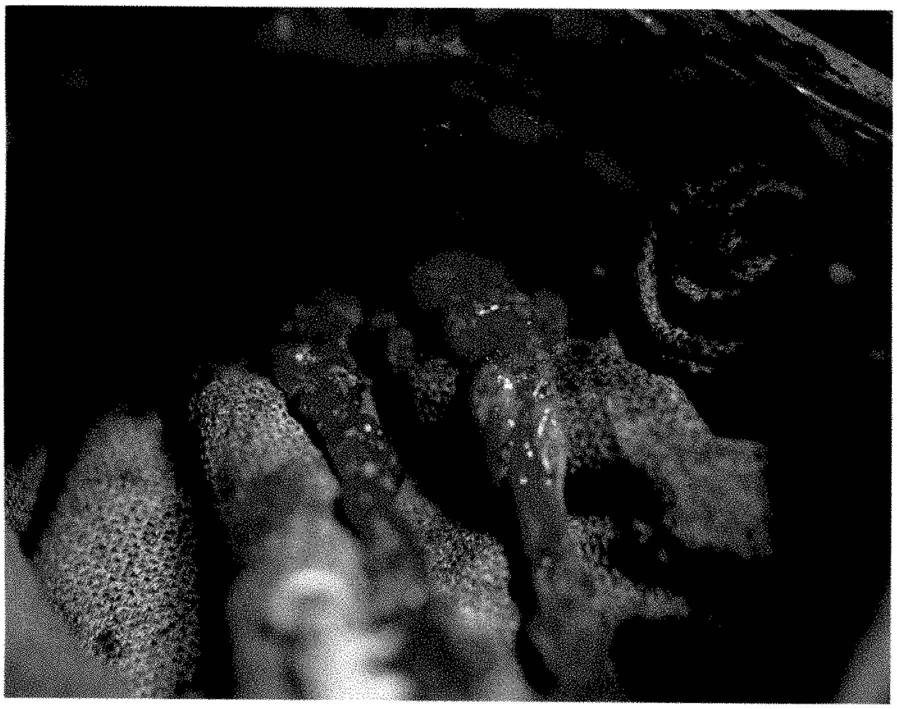

Fig 3. Lateral costal artery pedicle alongside the internal mammary artery pedicle of the right chest.

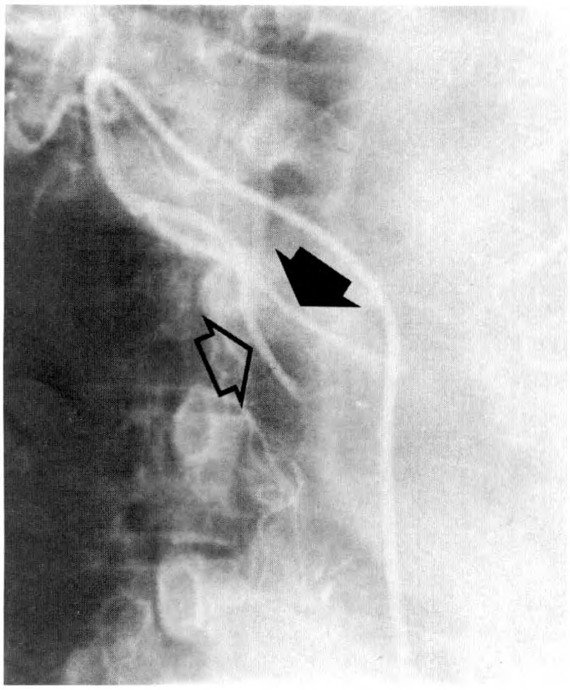

Fig 4. Selective injection of the right internal mammary artery (open arrow) *and right costal artery* (solid arrow). *The right costal artery is anastomosed to a diagonal branch.*

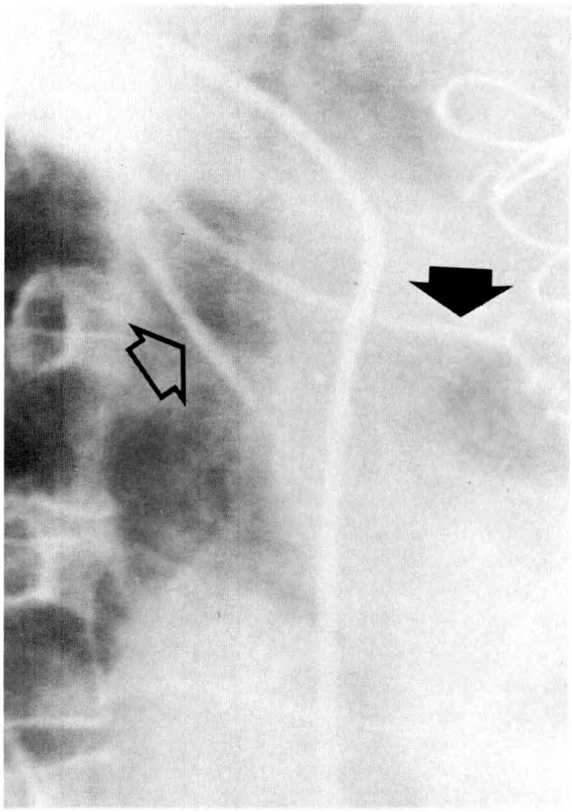

Fig 5. Selective injection of the right lateral costal artery (solid arrow) *and right internal mammary artery* (open arrow).

(Figs 4, 5). Histological examination of the distal ends of the lateral costal artery and internal mammary artery demonstrated no discernible difference.

Technique

The lateral costal artery is easily seen after the chest is retracted, as one prepares to harvest the internal mammary artery. Reducing the lung tidal volume compensated by increasing the ventilation rate adds considerably to the exposure. The artery is dissected from the chest wall as one would harvest the internal mammary artery, taking both veins and a broad pedicle containing muscle and fascia with the artery. The electrocautery blade is used as a spatula to separate the pedicle from the ribs and intercostal muscle. Arterial and venous branches are identified at each interspace and ligated with hemoclips. There is always a branch on each side of the pedicle. The lateral costal artery is harvested proximal to its junction with the internal mammary artery so as to allow complete mobility of the pedicle. We are injecting papaverine solution retrograde into the vessel as is done for the internal mammary artery to achieve maximal arterial dilatation.

Comment

The excellent patency rates reported for the internal mammary artery have fostered the trend toward complete myocardial revascularization with arterial conduits. Routinely using bilateral internal arteries can allow for two to four coronary arteries to be bypassed. The use of the gastroepiploic artery has allowed distal coronary lesions to be bypassed with an arterial conduit that hopefully will have the same long-term success as the internal mammary artery. The lateral costal artery is yet another arterial conduit that resembles the internal mammary artery, is very accessible, and hopefully will allow complete myocardial revascularization without resorting to saphenous veins.

Our initial experience demonstrates the feasibility of using the lateral costal artery for myocardial revascularization. Its diameter and histological appearance suggest that it can be viewed as an accessory internal mammary artery. Further study of the prevalence of this artery is needed, as well as long-term follow-up to determine the exact usefulness of this arterial bypass graft.

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Fatal Left Ventricular Perforation During Mitral Balloon Valvoplasty

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Percutaneous balloon dilation of left-sided cardiac valves is being performed with increasing frequency. We describe and document the mechanism of injury of left ventricular perforation during mitral balloon valvoplasty. Despite a technically successful surgical repair with hemodynamic restoration, the patient was brain dead. We emphasize (1) the risk and the potentially catastrophic nature of left ventricular perforation during mitral balloon valvoplasty and (2) the need for surgical standby and rapid operative decompression for this complication.

(Ann Thorac Surg 1990;49:819–21)

Successful mitral balloon valvoplasty for mitral stenosis was first reported by Inoue and associates [1] in 1984. This new technique has broadened the applicability of percutaneous catheter technology for stenotic valvular lesions and has provided an alternative to surgical procedures for patients who are high-risk candidates or refuse operation. The ultimate role of balloon dilation remains to be determined and will depend on its efficacy, durability, cost, and complication rate. We report a fatal case of left ventricular perforation during mitral balloon valvoplasty and document the cause.

A 65-year-old woman was admitted complaining of increasing dyspnea on exertion and limitation of activity for the past year. One year earlier, she had an episode of acute pulmonary edema due to mitral stenosis, which improved with digoxin and diuretics, and she refused mitral valve operation. On physical examination her blood pressure was 116/75 mm Hg and her heart rate was irregularly irregular at 94 beats/min. Auscultation of the heart revealed a prominent opening snap, a grade 1/6 diastolic rumble at the apex, and a grade 1/6 decrescendo blowing diastolic murmur at the left sternal border. The remainder of the examination was normal. The electrocardiogram showed atrial fibrillation and left atrial enlargement. A thickened, poorly mobile, and heavily calcified mitral valve was demonstrated by two-dimensional echocardiography and was consistent with severe mitral stenosis. Transseptal cardiac catheterization revealed a pulmonary artery pressure of 80/35 mm Hg, a mean left atrial pressure of 21 mm Hg, and a mitral valve area of 0.7 cm^2. A left ventricular angiogram showed no mitral regurgitation. Coronary arteriography was normal. The patient again refused an operation but agreed to mitral balloon valvoplasty. Investigational consent forms approved by the Human Subjects Committee were signed by the patient and her family.

With use of standard transseptal technique, a 0.97-mm (0.038 in.) guidewire with a large J was passed into the left ventricle. A balloon dilation catheter (7F with a 3-cm long, 8-mm wide balloon, Mansfield Scientific, Mansfield, MA) passed over the guidewire was used to dilate the atrial septum.

A second 0.97-mm guidewire was also positioned near the left ventricular apex through the repositioned transseptal sheath. A balloon dilation catheter (9F with a 20-mm wide, 5.5-cm long balloon) was passed over each guidewire and positioned across the mitral valve under fluoroscopic control. Both balloons were inflated simultaneously with a 50/50 mixture of contrast medium and 5% dextrose in water. During the first inflation, the "waist" of the stenotic mitral valve was located near the proximal end of each balloon. The balloons were repositioned more proximally for two further inflations, which resulted in loss of the "waist" in each balloon. Ten minutes after the last inflation, the blood pressure fell suddenly to 30 mm Hg systolic and the heart rate decreased to 30 beats/min. Intravenous fluids, blood, atropine, dopamine, and epinephrine were given and cardiopulmonary resuscitation was started. A two-dimensional echocardiogram was immediately performed and revealed a large pericardial effusion. Pericardiocentesis with a 7F pigtail catheter yielded 100 mL of blood. The blood pressure increased to 100 mm Hg systolic and the heart rate to 160 beats/min. However, severe hypotension recurred within ten minutes requiring aspiration of more blood with a transient improvement in blood pressure. At this time, an operating room that had not been on standby became available. At operation, several hundred milliliters of liquid blood and clot were removed from the pericardial space with rapid return of cardiac function. Two actively bleeding perforations of the left ventricular apex were closed with pledgetted mattress sutures. Although the patient was hemodynamically stable after operation, she never re-

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gained consciousness and serial electroencephalograms were consistent with brain death.

Comment

Since the original report by Inoue and associates [1] in 1984, mitral balloon valvoplasty has been adopted by many groups in the United States and abroad [2–7]. Valvoplasty was considered successful in 104 of 111 patients reviewed [3, 6–8]. The reported complications have included mitral regurgitation, atrial septal defect, groin hematoma, left atrial thrombus, thromboembolism, complete heart block, and perforation [9]. One death was reported in a patient who became hypotensive after failure to cross the mitral valve with the catheter. Despite emergency mitral valve replacement, this patient could not be weaned from cardiopulmonary bypass because of right heart failure [8].

Perforation as a complication of mitral balloon valvoplasty has been reported in 3 patients. In 1 patient, left ventricular perforation by the guidewire resulted in a small pericardial effusion, with fever as the only associated clinical problem [2]. In 2 patients, atrial wall injury led to termination of the procedure without further sequelae [7]. We report a fatal case related to perforation in the growing literature of mitral balloon valvoplasty. The mechanism of perforation is demonstrated by cineangiographic analysis. The two balloons were well positioned in the stenotic valve at the start of inflation as indicated by the balloon markers in Figure 1A. However, as inflation progressed, the balloons were squeezed out of the annulus and propelled through the left ventricular apex (Figs 1B, 1C). The guidewires, which were coiled in the left ventricle to protect against this injury, probably sliced through the thin left ventricular apex. This angiographic demonstration of the mechanism of injury correlates well with the two perforations found at operation.

In the absence of a pressure or volume load, as in isolated mitral stenosis, the left ventricle is small and thin and may be more prone to perforation during catheter manipulation. Furthermore, the left ventricle is a high-pressure chamber so that the risk of cardiac tamponade due to a perforation is increased. Before the era of balloon dilation of left sided cardiac valves, perforations at catheterization more commonly involved the lower pressure chambers. Whereas low-pressure cardiac chamber perforation may be managed with pericardiocentesis, left ventricular perforation has a greater potential for catastrophic tamponade and more often requires operative intervention.

Recognition of the risk of left ventricular perforation with balloon valvoplasty led to modifications in the technique of balloon dilation. Lock and co-workers [2] positioned the guidewire across the aortic valve with its tip advanced to the descending thoracic aorta. Despite using this technique, Lock and co-workers have reported 1 case of left ventricular perforation. Babic and colleagues [4] introduced transarterial balloon valvoplasty of mitral stenosis using a loop technique from the left femoral artery through the transseptal catheter to the right femoral vein. Theoretically the position of the balloon is better fixed by control of both ends of a long guidewire. However, the

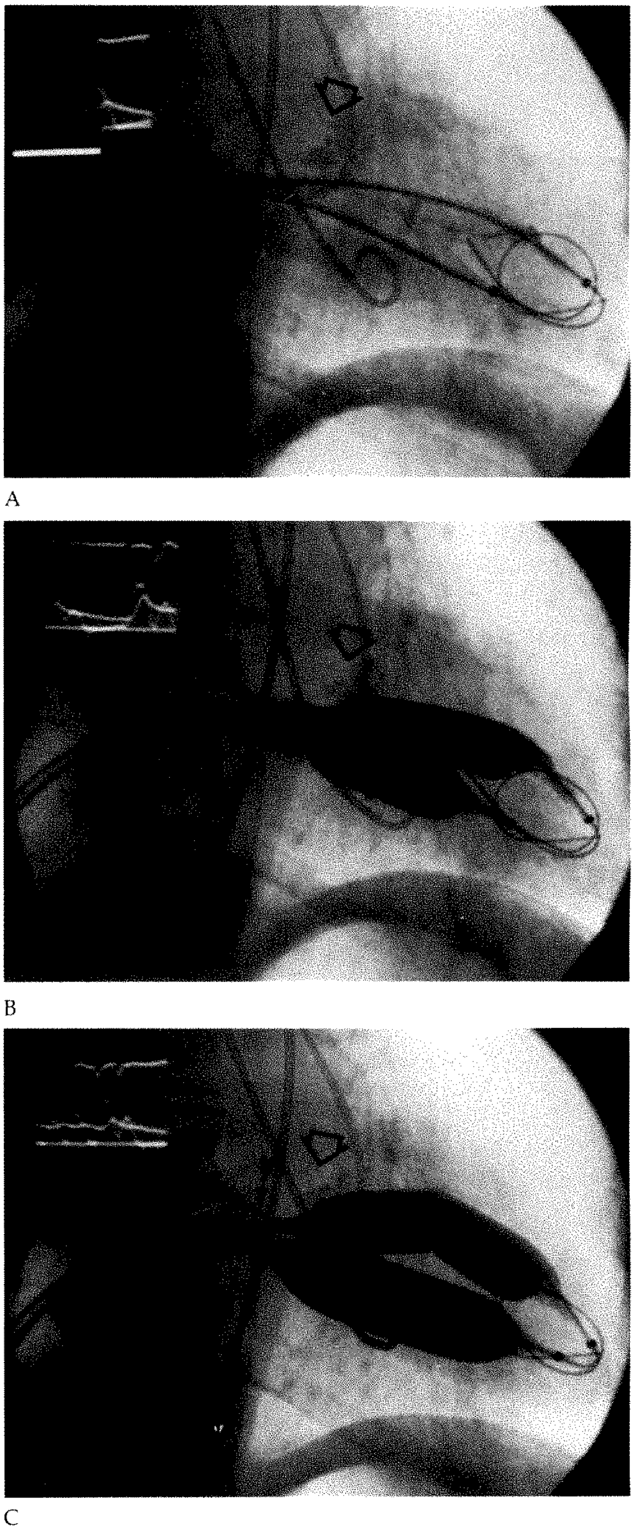

Fig 1. (A) The position of both balloons is demonstrated before inflation. The proximal end of one balloon is marked (solid arrow) *and can be compared with the plane of the mitral valve* (clear arrow) *which is visible as calcification. (B) At the beginning of inflation, the balloon waist is seen across the mitral valve. However, the balloons have begun to migrate into the left ventricle. (C) The balloons have been propelled into the left ventricle as evidenced by the loss of the balloon waist and the marked shortening of the distance between the proximal ends of the balloon markers* (solid arrow) *and the plane of the mitral valve* (clear arrow).

transarterial approach may be problematic in elderly patients with iliac artery disease.

Another technical consideration is the safety of using larger balloons or two simultaneous balloons. One study [5] showed that using larger or two simultaneous balloons further decreases the mitral valve gradient and increases the mitral valve area. It is unclear whether the two-balloon technique predisposed the patient to the injury that occurred.

The cost and time constraints of having an operating room and surgical team on standby have led many teams (including ours) to relax this practice, particularly because the incidence of complications requiring emergency operation has been low. However, in our case, the delay in operation probably contributed to the hypoxic brain damage. This catastrophic complication argues for having a surgical team on standby.

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Aortic Valve Disruption After Percutaneous Aortic Balloon Valvoplasty

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Severe disruption of the aortic valve cusps in patients with aortic valve stenosis can occur during percutaneous aortic balloon valvoplasty. We report such a case treated successfully by aortic valve replacement.

(Ann Thorac Surg 1990;49:822–3)

Percutaneous aortic balloon valvoplasty is used as an alternative to open heart operation for the treatment of patients with aortic stenosis. Initially introduced as a technique for treating patients who have been labeled as inoperable, the indications are being expanded to a wider population. Several groups are now advocating balloon valvoplasty as an alternative to operation in select patients with calcific aortic stenosis [1–4]. Evaluation of a new technique requires reporting of the procedure's morbidity and mortality. We report the case of a patient who had severe disruption of the aortic valve cusps during balloon valvoplasty requiring valve replacement.

A 75-year-old man was admitted to a hospital in Florida in December 1987 with an episode of syncope and a history of calcific valvular aortic stenosis. Cardiac catheterization showed an aortic valve gradient of 50 mm Hg, calculated aortic valve area of 0.53 cm^2, no aortic valve regurgitation, normal myocardial function, and single-vessel coronary artery disease. During that hospitalization, the patient underwent an aortic balloon valvoplasty complicated by transiently slurred speech. After valvoplasty, the aortic valve gradient decreased to 12 mm Hg, and the calculated aortic valve area increased to 1.0 cm^2. Ascending aortic angiography revealed 3+ aortic regurgitation.

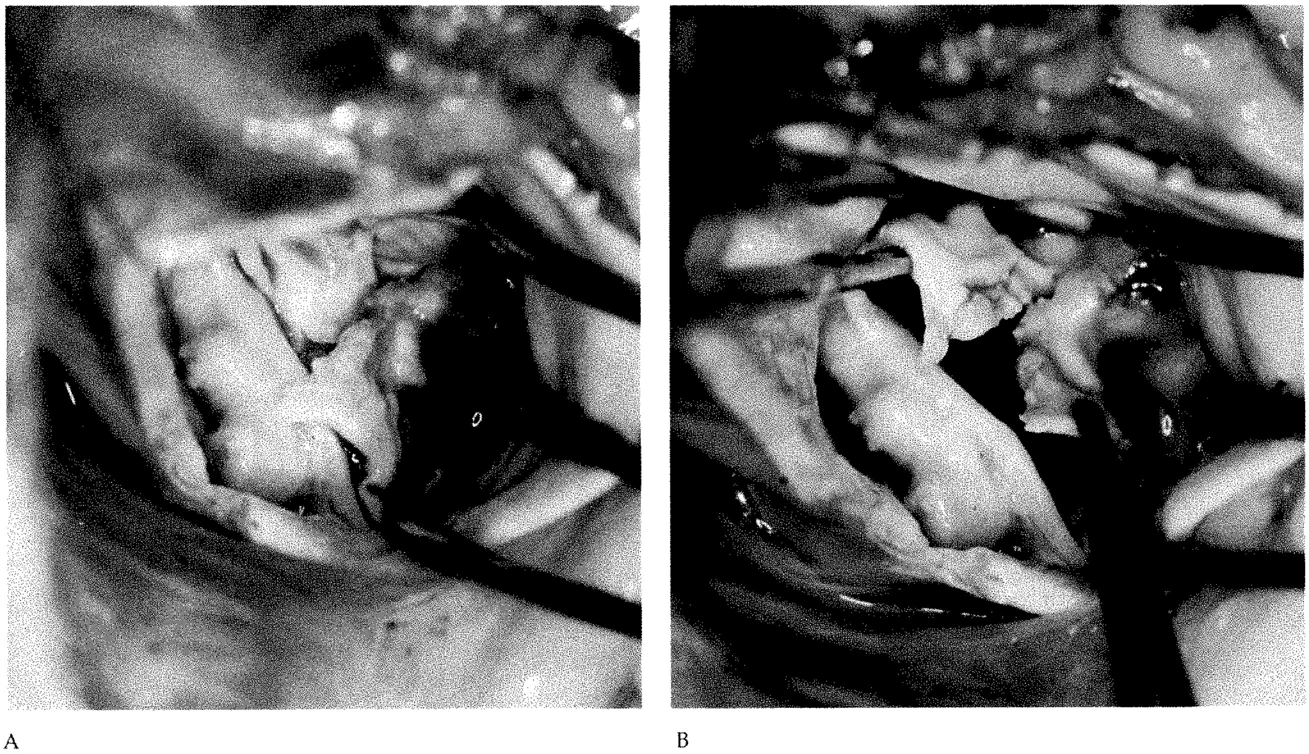

Fig 1. Intraoperative photos of aortic valve showing (A) cusp disruption and (B) extent of disruption between right and left leaflets.

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Over the ensuing 6 months, progressive shortness of breath and intractable congestive heart failure developed. The patient was referred to Deborah Heart and Lung Center, and he underwent repeat cardiac catheterization that showed an aortic valve gradient of 44 mm Hg, an aortic valve area of 0.6 cm^2, 4+ aortic regurgitation, and a severe left anterior descending coronary artery stenosis.

On May 23, 1988, the patient was taken to the operating room and the aortic valve was found to be heavily calcified with a complete tear of the right coronary cusp (Fig 1). The patient underwent aortic valve replacement with a 23-mm Carpentier-Edwards tissue valve and a bypass graft to the left anterior descending coronary artery. The patient had a normal convalescence with an uneventful postoperative course.

Comment

Currently revived as a nonsurgical procedure, percutaneous aortic balloon valvoplasty is gaining increased acceptance. Conceptually, there is no reason to expect the results obtained with percutaneous valvoplasty to be superior to those obtained with closed surgical techniques. Initial reports confirm that the results obtained with percutaneous valvoplasty are generally far from satisfactory, and many complications including aortic valve injury have been documented [4–7].

Percutaneous valvoplasty may be indicated in a select group of moribund patients, but currently, it should not be considered an alternative to operation. This patient was a low surgical risk, but was assigned by his cardiologist to undergo percutaneous balloon valvoplasty because of the prevailing attitude that percutaneous valvoplasty procedures are simple to perform, carry a low risk, and are as effective as established surgical procedures. The procedure was performed at considerable expense and resulted in morbidity and an unsatisfactory result. The complications associated with this attempt to avoid operative intervention should prompt physicians to question the merit of this new procedure.

Despite encouraging preliminary results, percutaneous balloon valvoplasty has inherent risk and should be used only in select patients. We report, however, the successful surgical management of cusp disruption secondary to percutaneous balloon valvoplasty.

We gratefully acknowledge Alison Perritt, RN, BSN, Donna Mull, and Michelle Pallante for their help in preparation of the manuscript.

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Surgery of Giant Bulla With Tube Drainage and Bronchofiberoptic Bronchial Occlusion

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A case of emphysematous bullae and right lung cancer is presented. At first, left giant bulla was managed by minithoracotomy and tube drainage combined with bronchofiberoptic bronchial occlusion to preserve the respiratory function. Four weeks later, right lung cancer was successfully resected.

(Ann Thorac Surg 1990;49:824–5)

For surgical treatment of giant bulla, limited "tailored" resections such as plication-excision, Naclerio and Langer's method, or bullectomy with stapling devices are widely adopted [1–3]. We developed a new method consisting of intracavitary suction under minithoracotomy and endobronchial occlusion under a bronchofiberscope. The operative procedure and its advantages are described.

A 72-year-old man with a 3-year history of bilateral emphysematous bullae was admitted with a right abnormal shadow discovered anew on the chest x-ray film. A computed tomographic scan showed a right lung tumor mass and bilateral multiple bullae, especially a marked giant bulla occupying the lower half of the left hemithorax and compressing the diaphragm (Fig 1). His pulmonary function and arterial blood gas analysis data were as follows: forced vital capacity, 2.39 L; forced expiratory volume in 1 second, 0.69 L (28.8%); arterial oxygen tension, 66.2 mm Hg; and arterial carbon dioxide tension, 48.5 mm Hg.

Because of the poor pulmonary function, the question was how to perform the operation safely. To get the pulmonary function restored with minimal loss, operation for the left giant bulla was performed. With the patient in the lateral position under spontaneous breathing and epidural anesthesia, a small left thoracotomy was performed through the ninth intercostal space. The length of the skin incision was about 5 cm. The wall of the bulla was held by forceps and a stay suture was placed. A bronchofiberscope was inserted into the bulla, then fibrin glue and OK-432 (immunoactivator that is also commonly used as a tissue adhesive in Japan) was injected into the bottom of the bulla. A drain was placed in the bulla and fixed to the chest wall using a stay suture. With suction and the complete collapse of the bulla, artificially created pneumothorax was managed by thoracic drain. The air in the bulla was drawn with 20-cm H_2O underwater seal drainage. The thoracic drain was attached to 5-cm H_2O underwater seal drainage. The wound was closed by serratus anterior muscle suture and skin closure.

After the previously mentioned intracavitary suction procedure, the patient was made to lie on his back and bronchofiberscopy was performed under surface anesthesia. A bronchial occlusion test with Fogarty's balloon catheter showed that occlusion of the B10 bronchus led to decrease of the air leakage from the bulla. To keep the two components of the fibrin glue from mixing and reacting before they reached the segmental bronchus, we used a 16-gauge double-lumen central venous catheter inside the channel of the bronchofiberscope. The two components of the glue were injected simultaneously from two syringes into the B10 bronchus.

Soon after the operation, the subjective symptoms of the patient improved. The air leakage from the bulla ceased on the third postoperative day. To make it possible for the patient to walk, the continuous underwater seal drainage was replaced by a portable wound suction unit, which was removed on the fifth postoperative day. Data for the pulmonary function and arterial blood gas analysis on the seventh postoperative day were as follows: forced vital capacity, 2.61 L; forced expiratory volume in 1 second, 1.53 L (58.6%); arterial oxygen tension, 66.4 mm Hg; and arterial carbon dioxide tension, 40.5 mm Hg.

Postoperative computed tomographic scan showed a satisfactory result (Fig 1). Resection of the right lung cancer and right pneumorrhaphy was performed uneventfully 4 weeks after the first operation. No recurrence of bullae has been recognized in the 10-month follow-up period.

Comment

Operation for giant bulla is widely accepted to relieve disabling dyspnea and to improve pulmonary function. Procedures such as plication-excision, Naclerio and Langer's method, or bullectomy with stapling devices are widely adopted, but they have some problems, including loss of the pulmonary function caused by thoracotomy itself, weaning from mechanical ventilation after general anesthesia, and protractive postoperative alveolar fistula.

The intracavitary suction procedure is a classic method [4] that has the following advantages: (1) the operation is

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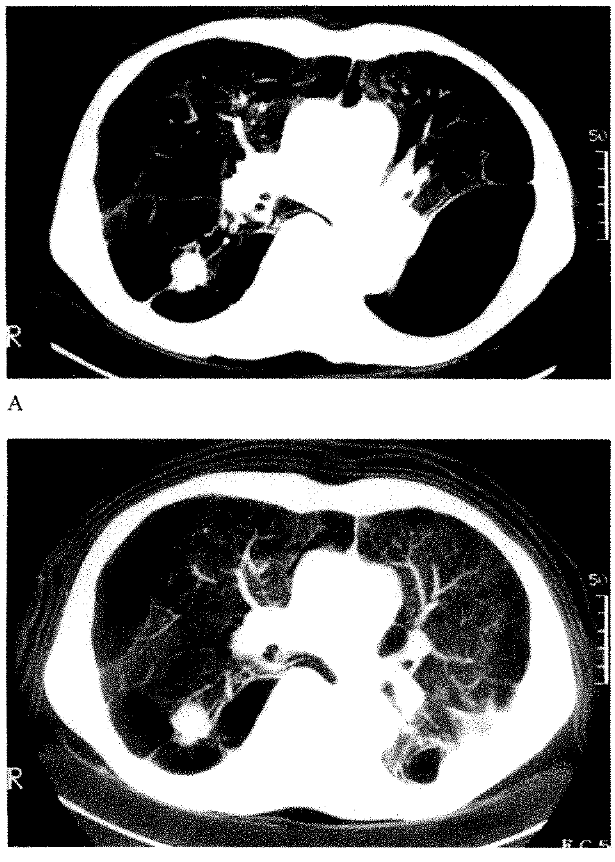

Fig 1. (A) Computed tomographic scan demonstrating a giant left bulla and a right lung cancer adjacent to medium-sized bullae. (B) Complete collapse of giant left bulla with cicatrization and expansion of the left lower lobe (adjacent small bulla is remaining).

easy and can be performed in a short time, (2) positive-pressure ventilation, which may produce enlargement of contralateral bullae, is not needed, (3) adjacent lung tissue is not sacrificed, and (4) the risk is less. However, it is chosen at only a few centers at present [5]. The reason why this procedure has not been popularized seems to be that the drainage of air from the bulla is massive and protractive and that the bulla is diminished but not eliminated completely in some cases.

Our procedure was developed to reduce these disadvantages. Recently postoperative alveolar leakage and intractable pneumothorax has been successfully treated by bronchial occlusion without any complications [6, 7]. Temporary occlusion of the drainage bronchus with fibrin glue makes it possible to reduce the air leakage from the bulla, which makes the continuously drained cavity as small as possible.

Pneumothorax might be a problem with this procedure. However, with suction of the intrabullous space and an only momentary pneumothorax, intrathoracic air does not exceed the intrabullous air volume that had already been reduced. We think it would help the bulla to completely collapse but would not sacrifice the pulmonary reserve. The intrabullous drain must have greater suction than the intrathoracic one.

Many kinds of adhesive agents, such as minocycline, BCG, and OK-432, have been developed and used to treat postoperative alveolar leakage or the intractable pneumothorax. Intracavitary administration of them will lead the bullae to diminish with cicatrization in the early phase.

In this case, a marked improvement in pulmonary function was obtained on the seventh postoperative day. On closure of the small intercostal thoracotomy, which was about 3 cm, only serratus muscle and skin were sutured, which caused minimal chest pain. Recently, we have successfully applied this method to giant bulla in an 82-year-old man with dyspnea at rest. In both cases the air from the bulla stopped on the third postoperative day. This is substantially shorter than the period of seven days or more taken by intracavitary suction only [4, 5].

The combination of intracavitary suction, endobronchial occlusion, and intracavitary administration of tissue adhesive agents has many advantages, and may offer an attractive alternative in treating patients with giant bulla.

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Use of Bovine Lung Heparin to Obviate Anaphylactic Shock Caused by Porcine Gut Heparin

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Intracardiac repair in a 7-year-old girl with tetralogy of Fallot was hampered because of an anaphylactic reaction with cardiopulmonary collapse caused by porcine gut heparin. This patient had an extremely rare case of hypersensitivity to porcine gut heparin but not to bovine lung heparin. Uneventful radical operation could be performed with the use of bovine lung heparin for systemic heparinization during cardiopulmonary bypass.

(Ann Thorac Surg 1990;49:826–7)

Heparin anaphylaxis with cardiopulmonary collapse is a rare complication of cardiac operations. This hypersensitivity is attributed to the species of heparin, duration of heparin use, and antigenic heterogeneity of heparin lots. In this study, we present an extremely rare case of anaphylactic shock induced by porcine gut heparin but not by bovine lung heparin.

A 7-year-old girl weighing 16.5 kg was referred to Sakakibara Heart Institute for intracardiac repair of tetralogy of Fallot. She underwent Brock's operation and ligation of the patent ductus arteriosus at another hospital when she was 7 months old. Preoperative cardiac catheterization on December 9, 1987, demonstrated typical tetralogy of Fallot and intracardiac repair was proposed. On August 2, 1988, anesthesia was induced by the intramuscular injection of ketamine (85 mg). Then pancuronium bromide (3 mg) was administered intravenously and an endotracheal tube was placed without complication. In routine fashion, an 18-gauge catheter was introduced into the right radial artery for continuous blood pressure monitoring and flushed with a couple of milliliters of heparinized saline solution (2 mg heparin in 500 mL of saline solution). Immediately thereafter, blood pressure decreased abruptly from 130/90 to 60/0 mm Hg. Swelling and redness developed on the right palm, right upper arm, and right anterior chest. Metaraminol, methylprednisolone, and epinephrine were required to maintain systolic blood pressure over 100 mm Hg. Anaphylactic shock was presumed, but the allergen was unknown at that time. The operation was suspended and the patient was transferred to the intensive care unit. She recovered from anaphylaxis with appropriate medical therapy.

Two months after this event, ketamine, pancuronium bromide, and heparin were evaluated to determine whether one of them was the allergen for anaphylactic shock. However, the lymphocyte-stimulating test demonstrated that none of these drugs was the allergen. Although heparin remained under suspicion, all three drugs were excluded as possibilities.

Paying special attention to the use of heparin, we decided to perform a radical operation on March 14, 1989. A slow induction technique using nitrous oxide, oxygen, and ethylene was used for anesthesia. After the intravenous administration of pancuronium bromide (2 mg), the endotracheal tube was introduced. Then, an 18-gauge catheter was introduced into the right radial artery for continuous blood pressure monitoring and heparinized saline solution was flushed through very carefully. Again, the patient became flushed and her blood pressure decreased from 130/90 to 80/0 mm Hg. Swelling and redness developed on the right palm, right upper arm, and right anterior chest. Careful observation revealed a raised erythematous rash on the thumb, index finger, and middle finger of the right hand that corresponded to the area fed by the right radial artery. The compliance of the lung deteriorated immediately, resulting in severe pulmonary congestion. Forced manual ventilation with 100% oxygen was required to maintain adequate arterial oxygen tension. Epinephrine was administered intravenously to maintain systolic blood pressure over 100 mm Hg. Thirty minutes later, the patient had recuperated from pulmonary congestion and systemic hypotension. It was confirmed that the anaphylactic reactions were induced by the administration of heparin into the right radial artery.

Because porcine gut heparin had been used up to that time, bovine lung heparin was considered the only recourse. Bovine lung heparin was tested to determine whether it induced an anaphylactic reaction. Five milliliters of diluted bovine lung heparin (2 mg heparin in 500 mL of saline solution) was infused into the catheter in the right radial artery, but no anaphylactic reaction occurred.

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The operation was suspended because of the patient's unstable general condition. Three days after the second episode of anaphylactic shock to porcine gut heparin, intracardiac repair was performed uneventfully with the use of bovine lung heparin throughout the operation. The patient was discharged from the hospital without any complications.

Comment

With the development of purified forms of heparin, anaphylaxis with cardiopulmonary collapse has become a rare complication of its use. The frequent anaphylactic reactions noted during the early use of heparin are now known to have resulted from allergic response to preservatives rather than to the drug itself [1–3]. Because highly purified forms of heparin are used at the present time, heparin allergy is rare, and only a few cases of heparin anaphylaxis or hypersensitivity have been reported in the English-language literature [4–7]. Furthermore, specific allergic reactions depending on the species of heparin (porcine versus bovine) are extremely rare. Rosenzweig and associates [4] reported a hypersensitivity reaction to porcine heparin but not to bovine heparin in a patient during the course of acute renal failure. Their immunological studies suggested that the allergic response was to a contaminant of the heparin and involved both immunoglobulin G and immunoglobulin E antibodies. In the present patient, we confirmed the anaphylactic reaction to porcine gut heparin because of the reproducibility of anaphylaxis after infusion of heparinized saline solution into the right radial artery. The patient seemed to be sensitized to porcine gut heparin when she underwent cardiac catheterization 9 months previously. The porcine gut heparin used in our patient was purified from porcine gut, and the other ingredients were saline solution and benzyl alcohol. The bovine lung heparin was purified from bovine lung, and the other ingredients were again benzyl alcohol and saline solution. There was no difference in preservatives between the two heparins. It is possible that the allergen in our patient was not the heparin moiety itself or the preservatives but a contaminant, presumably of gut origin, in the porcine preparation.

Immunological examinations including the lymphocyte-stimulating test failed to prove that porcine gut heparin was the allergen in our patient. It is possible that an anaphylactic reaction occurred in vivo, despite the failure to demonstrate a specific reaction in in vitro immunological examinations.

Although the patient was not hypersensitive to bovine lung heparin, she may become sensitized to it because she was exposed to bovine lung heparin at the time of the second anaphylaxis. Therefore, we decided to perform intracardiac repair only three days after the second anaphylaxis, before sensitization to bovine lung heparin could be completed.

In conclusion, based on our experience, we would advocate using bovine lung heparin in patients with anaphylaxis induced by porcine gut heparin, and in contrast, using porcine gut heparin in patients with anaphylaxis induced by bovine lung heparin.

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Cystic Pulmonary Hamartoma

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A case of cystic pulmonary hamartoma is reported and the literature is reviewed. According to the literature, only 6 well-documented cases have been previously reported, and all the patients including our patient fared well after operation.

(Ann Thorac Surg 1990;49:828–9)

Cystic pulmonary hamartomas are very rare. We encountered a patient with a cavitary lesion that was diagnosed as cystic pulmonary hamartoma after resection. We describe the case in this report.

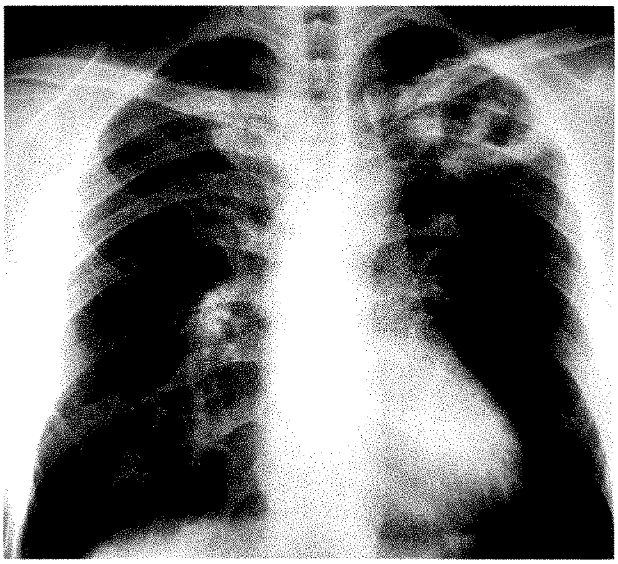
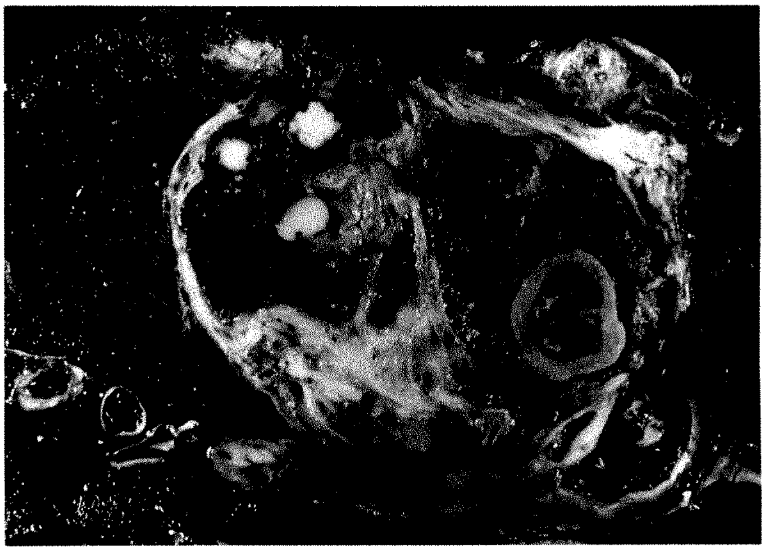


Fig 1. Posteroanterior chest roentgenogram showing cavitary lesion in the left upper lobe.

A 49-year-old man diagnosed as having pulmonary tuberculosis for 2 years was referred to our hospital. The results of physical examination and laboratory studies were normal, and the results of sputum smears and cultures were negative. Chest roentgenogram (Fig 1) showed a cavitary shadow on the left upper lobe. The patient underwent left upper lobectomy with the diagnosis of pulmonary tuberculosis. The patient had an uneventful postoperative course. The cut surface (Fig 2) showed a well-circumscribed 7 × 6-cm tumor that had epithelial-lined cavities containing nodular aggregate of firm gray-white tissue. Microscopically, the tumor was composed of bronchiolar structures lined with ciliated columnar epithelium, adipose tissue, cartilage, smooth muscle, neural tissue, and bone marrow (Fig 3). The pathological diagnosis was cystic hamartoma.

Fig 2. Cut surface showing the tumor, which has cavities.



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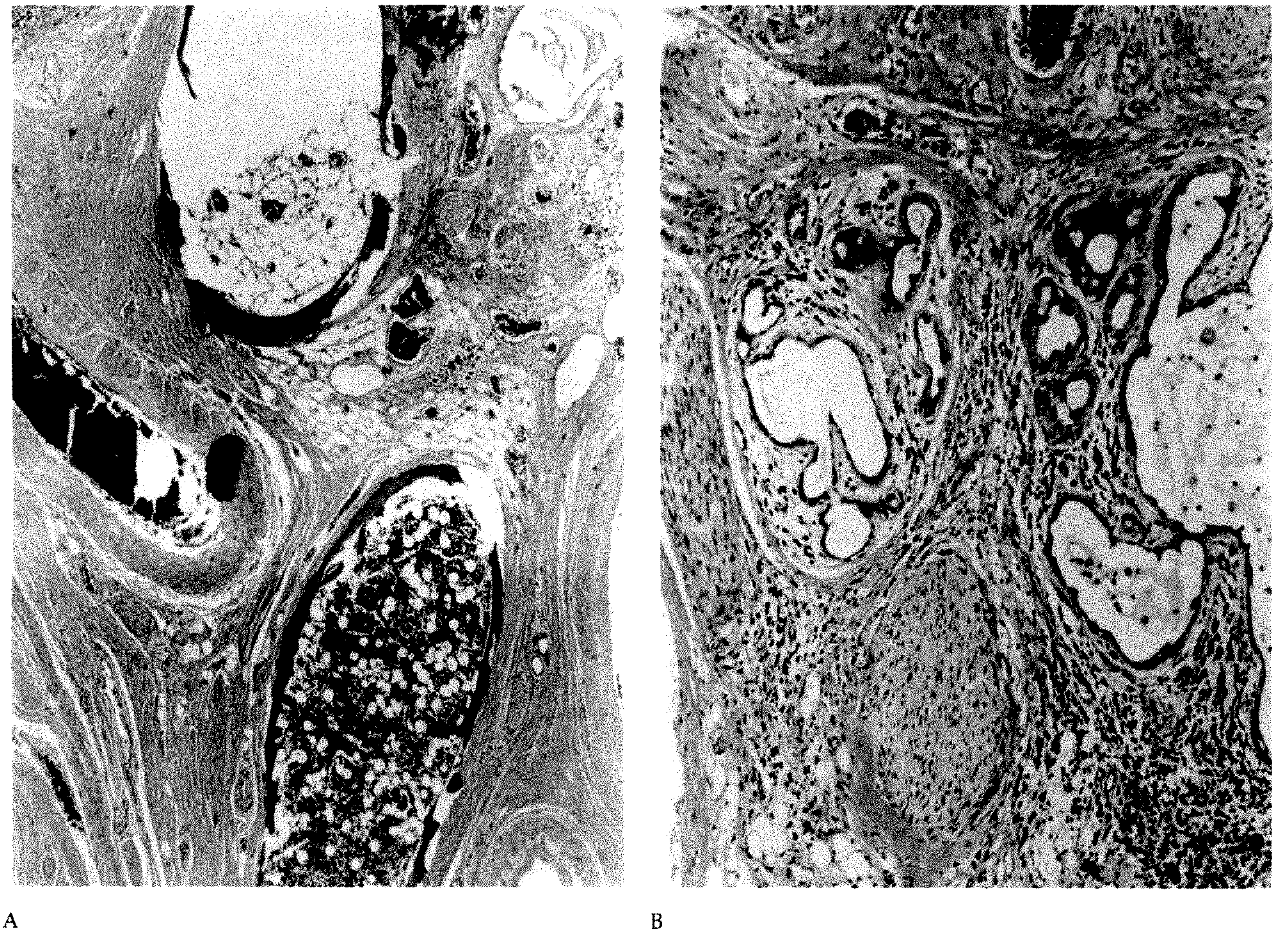

Fig 3. The tumor is composed of bronchiolar structures lined with ciliated columnar epithelium, adipose tissue, cartilage, smooth muscle, neural tissue, and bone marrow. (Hematoxylin and eosin, ×10 before 32% reduction.)

Comment

Hamartoma is the most common benign neoplasm of the lung, and accounts for 7% of all solitary lung nodules [1]. Cystic pulmonary hamartomas, however, are very rare. Only 6 cases have been previously reported, 3 female patients and 4 male patients (median age, 45 years) [1–5]. Four patients were asymptomatic at diagnosis, and 3 patients had dyspnea and hemoptysis. Six of the 7 patients had the lesion in the left lung, but the radiographic appearance of this type of hamartoma is not specific. No cases were reported diagnosed as cystic pulmonary hamartoma before operation. These patients were misdiagnosed as having tuberculosis, echinococcus cyst, congenital or acquired cyst, dermoid cyst, teratomatous cyst, or bronchogenic carcinoma. A tissue examination of the resected lesion was required to establish the diagnosis. The prognosis after surgical excision was excellent.

The mechanism of cyst development within a hamartoma is unknown. The route of entry of air into these lesions can only be hypothesized [3]. A check-valve mechanism might result in the gradual expansion of small epithelial-lined tubules resembling bronchioles, but no bronchial connection to the air-filled multicystic area was found in our patient, and none have been reported in cystic hamartomas. Jensen and Schiødt [6] have described the growth conditions of pulmonary hamartomas; we assume that the cleftlike spaces of pulmonary hamartomas expand to become growing cysts.

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Recurrence and Malignant Transformation of Endotracheal Chondroma

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An endotracheal chondroma with recurrent course and malignant transformation is described. The first chondroma recurrence appeared 5 years after the primary operation, with no histopathological signs of malignancy. A second recurrence after a further 6 years showed obvious histological evidence of malignant chondrosarcoma. A third recurrence after 1 more year metastasized to various organs and led to the patient's death 14 years from the date of primary diagnosis.

(Ann Thorac Surg 1990;49:830–2)

Primary and especially cartilaginous tumors of the trachea are very rare [1, 2]. Only a few cases of chondromas, hamartomas, and chondrosarcomas have been reported [3–7]. Reviewing the literature, we found only 1 case of recurrent chondroma [4] and none of malignant transformation of benign chondroma to chondrosarcoma.

A 57-year-old man had been under medical care for diabetes for 10 years. He suffered first from cough and dyspnea for some weeks and in April 1967, from high fever. Three months later, the symptoms recurred, and wheezing, hemoptysis, and profuse productive cough developed. Chest roentgenogram showed no parenchymal infiltration.

The patient was further evaluated. At bronchoscopy, a large tracheal tumor 25 cm from the upper teeth was found. After the bronchoscopy, an operation was carried out. A right-side thoracotomy was performed, and the trachea was exposed. A 3 × 3-cm tumor was seen in the anterior wall of the trachea above the bifurcation. One third of the tumor was growing outside the trachea, and two thirds seemed to be inside it (Fig 1). The tumor was removed, leaving only the thin outer capsule—a bulging wall covered by a pleural flap. The patient recovered uneventfully.

Histological examination showed a cartilaginous neoplasm consistent with benign chondroma (Fig 2). The density of chondrocytes was modest and even. Their nuclear atypia was slight, and no increased mitotic rate was observed. There were thus no signs of malignancy.

The patient was examined postoperatively twice a year until May 1972. He was asymptomatic throughout this period. In autumn 1972, dyspnea again developed in the patient. A bronchoscopy in December showed an occluding tumor 25 cm from the upper teeth. The second right thoracotomy was done. The tumor, about 6 cm in diameter, was growing endotracheally but mostly extratracheally. The trachea was severed 2 cm above the bifurcation and 5 cm of the trachea was resected. The reconstruction was done with end-to-end anastomosis. The patient recovered uneventfully.

Histological examination of the surgical specimen confirmed the diagnosis of a benign chondroma without signs of malignancy (Fig 3). The microscopic picture was comparable with that of the previous specimen with the exception of increased cellularity.

Several control bronchoscopies were done without evidence of recurrence or stricture. In summer 1978, almost 11 years after the first operation, a mediastinal tumor was suspected based on the chest roentgenogram of the patient, now 68 years old. In August 1978, the third right thoracotomy was done; a large tumor, about 10 cm in diameter, was seen in the mediastinum. The tumor was removed together with a piece of the pericardium and lung, in which the tumor had infiltrated.

At microscopy there was an evident malignant chondrosarcoma. Chondrocytes and chondroblasts had increased in number, showing pronounced nuclear atypia and mitotic figures.

The patient recovered, but 1 year later a fast-growing tumor appeared on the right side of the chest wall. In January 1980, the sixth to eighth ribs were partially resected. Two tumors were infiltrating the rib cartilage; histologically, they were metastatic chondrosarcomas. In February 1981, a recurrent tumor about 3 cm in diameter was growing into the eighth rib cartilage. The tumor was excised and examined. It was also metastatic chondrosarcoma.

In June 1981, the patient had right hemiplegia. A cerebral metastasis was diagnosed by computed tomography and the patient died in August 1981, almost 14 years after the first tracheal operation and 3 years after the tumor had been verified as malignant. Postmortem examination showed a metastasis in the left cerebral hemisphere. The right lung had a 10 × 15-cm tumor and an abscess next to it. Some small metastases were found in the left lung, one in the liver, and one also in each kidney.

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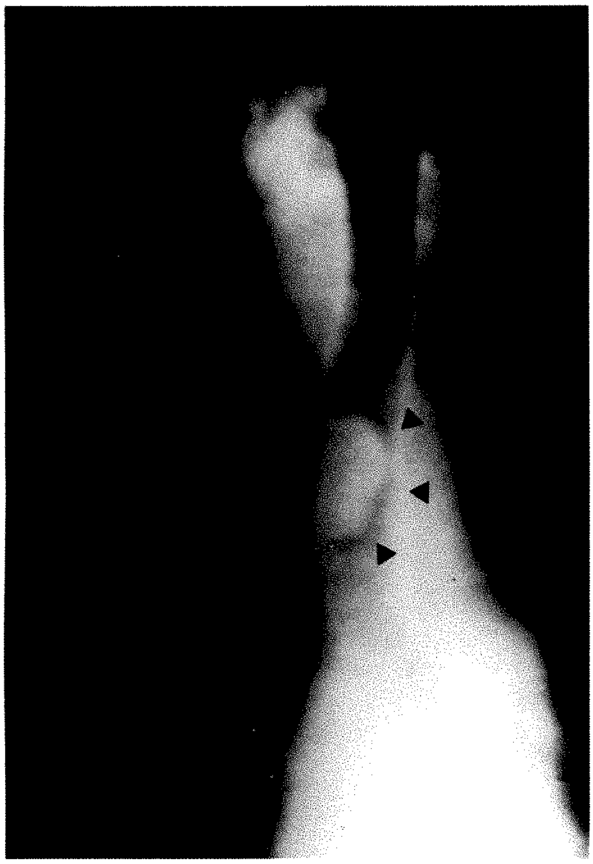

Fig 1. Tracheal tomogram, showing the primary tumor inside the trachea.

All these infiltrates were histologically examined and verified as metastatic growths of chondrosarcoma (Fig 4).

Comment

Due to their low frequency, benign and malignant tumors of the upper airways are diagnostic problems. Such a tumor is often asymptomatic until it occludes 60% to 75% of the tracheal lumen [1, 3]. Because the common symptoms are cough, stridor, and dyspnea, these neoplasms are often misdiagnosed as asthma [1, 3, 8, 9]. The tumor can only be diagnosed with certainty by bronchoscopy. Benign tumors have been bronchoscopically removed [3, 8, 9], but curative resection with adequate margin of normal tissue must be recommended for treatment of both the benign and malignant cartilaginous neoplasms of the trachea because of their tendency to recur [3, 4].

In our patient, the primary tumor was interpreted as benign and encapsulated. At the removal of the tumor, a thin capsule was left to form tracheal wall. No tracheal resection was performed. This operation allowed a 5-year asymptomatic period; however, the tumor recurred. At the second operation, it was still considered a typical benign chondroma. The tumor was radically removed,

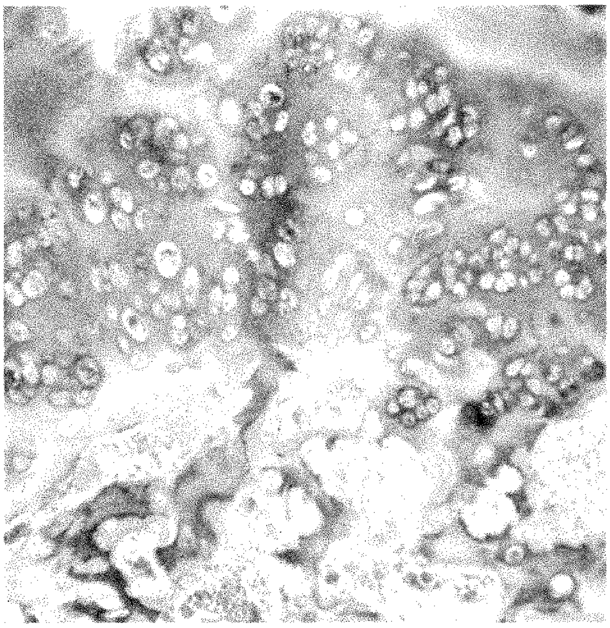

Fig 3. The tumor recurrence is a benign chondroma with increased cellularity. (Hematoxylin and eosin, ×1,000 before 36% reduction.)

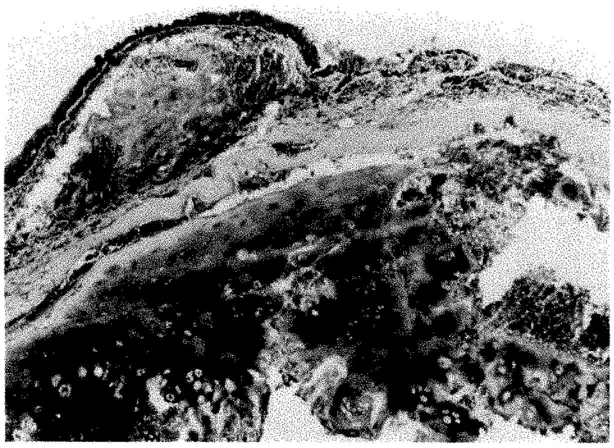

Fig 2. The primary tumor is cartilaginous and consistent with benign chondroma. (Hematoxylin and eosin, ×500 before 54% reduction.)

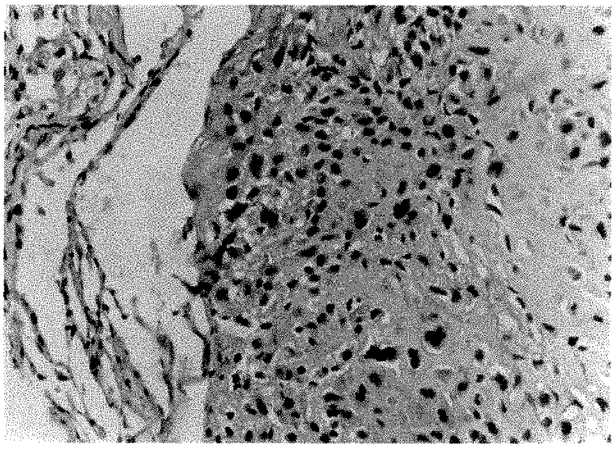

Fig 4. The pulmonary metastasis is an evident malignant chondrosarcoma. (Hematoxylin and eosin, ×1,200 before 54% reduction.)

and a 5-cm tracheal resection was done. About 6 years later, the tumor recurred again and transformed to histologically typical chondrosarcoma. The recurrence and further malignant transformation of the neoplasm could possibly have been avoided with radical tracheal resection at the first operation. Some authors [4, 6] have mentioned the potential of chondroma to undergo sarcomatous change, but this has not been well documented. On the other hand, this recurrence after resection of the trachea raises the question of multifocal origin of the tracheal cartilaginous neoplasms.

It may be difficult to classify chondrosarcomas, because the histopathological criteria for malignancy are poor [2, 3]. In our patient, however, the primary tumor was without any malignant characteristics. Its second recurrence, diagnosed as chondrosarcoma, had obvious malignant features, ie, increased cellularity and mitotic rate, as well as infiltrative growth of the neoplastic tissue in histological examination.

Because only a few tracheal chondromas have been described, the potential for malignant change should be kept in mind for their treatment. Accordingly, we strongly prefer radical removal of the tumor with curative tracheal resection to ensure that the anastomosis is tumor-free.

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Notice From the American Board of Thoracic Surgery

The part 1 (written) examination will be held at the Hyatt-Regency, Dallas Fort Worth Airport, Dallas TX, on February 3, 1991. The closing date for registration is August 1, 1990.

To be admissible for the part II (oral) examination, a candidate must have successfully completed the part I (written) examination.

A candidate applying for admission to the certifying examination must fulfill all the requirements of the board in force at the time the application is received.

Please address all communications to the American Board of Thoracic Surgery, One Rotary Center, Suite 803, Evanston, IL 60201.

Circumflex Exposure Using a Cardiac Sling

I. F. Galvin, FRCSI, and D. C. Newman, FRACS

Department of Cardiothoracic Surgery, The Prince Henry Hospital, Sydney, Australia

A highly efficient and technically useful method of circumflex coronary artery exposure using a cardiac sling is described.

(Ann Thorac Surg 1990;49:833–4)

Most surgeons find the marginal branches of the circumflex coronary artery among the most difficult to expose for anastomosis. These arteries, lying as they do on the lateral and posterior aspect of the heart, are only well visualized when skilled assistance is available. This usually means an assistant standing to the surgeon's left retracting the heart laterally. This assistant cannot see what he or she is doing, often gets in the surgeon's way, and may lose traction on the heart at a vital moment. We describe a technique used at our hospital that markedly improves exposure for circumflex anastomoses.

Technique

One half of a standard 36 × 4-inch (91 × 10-cm) Raytec gauze strip is dipped in saline solution. The dry half of the gauze is looped, singly knotted, and fixed to the right side of the chest retractor. The lateral aspect of the oblique sinus above the inferior vena cava is opened with a finger and the wet end of the other extremity of the gauze strip is fed through the sinus to the left of the heart with a Semb Ligature carrier (Fig 1A). The sling is now ready for use, and when the appropriate time for a circumflex anastomosis is reached the heart is grasped within the substance of the surgeon's right hand, apex toward the heel of the wrist, thumb gently depressing the right ventricular body inward while the fingers fan outward over the lateral convexity of the heart (Fig 1B). The hand is now rotated gently in a counterclockwise manner making the obtuse margin of the heart present uppermost. While performing this maneuver the gauze sling, the end of which is in the surgeon's left hand, is gently used to retract and fix the heart by securing the gauze to the right of the chest retractor with a towel clip. This now gives fixed presentation and excellent exposure of the obtuse marginal artery to the operator (Fig 1C). When experience and familiarity with this technique are achieved any region of the lateral or posterior left ventricular wall is accessible. To expose a distal marginal or a posterior descending coronary artery, bifurcation and repositioning of the sling is all that is required (Figs 1D–F). We have found that as well as obviating the need for a second assistant, these methods of retraction and exposure afford optimum conditions and make for easy and expeditious anastomoses.

Comment

We have used this method of cardiac retraction since 1984. The technique can be used in conjunction with single two stage cavoatrial or bicaval cannulation but cannot be employed when a right atrial basket is used for venous return. We have noted with interest the contributions made by Janke [1] and more recently Angelini [2] in the area of coronary exposure, but feel that our sling is an efficient, easily applied, inexpensive technical aid for exposure of the circumflex coronary artery branches.

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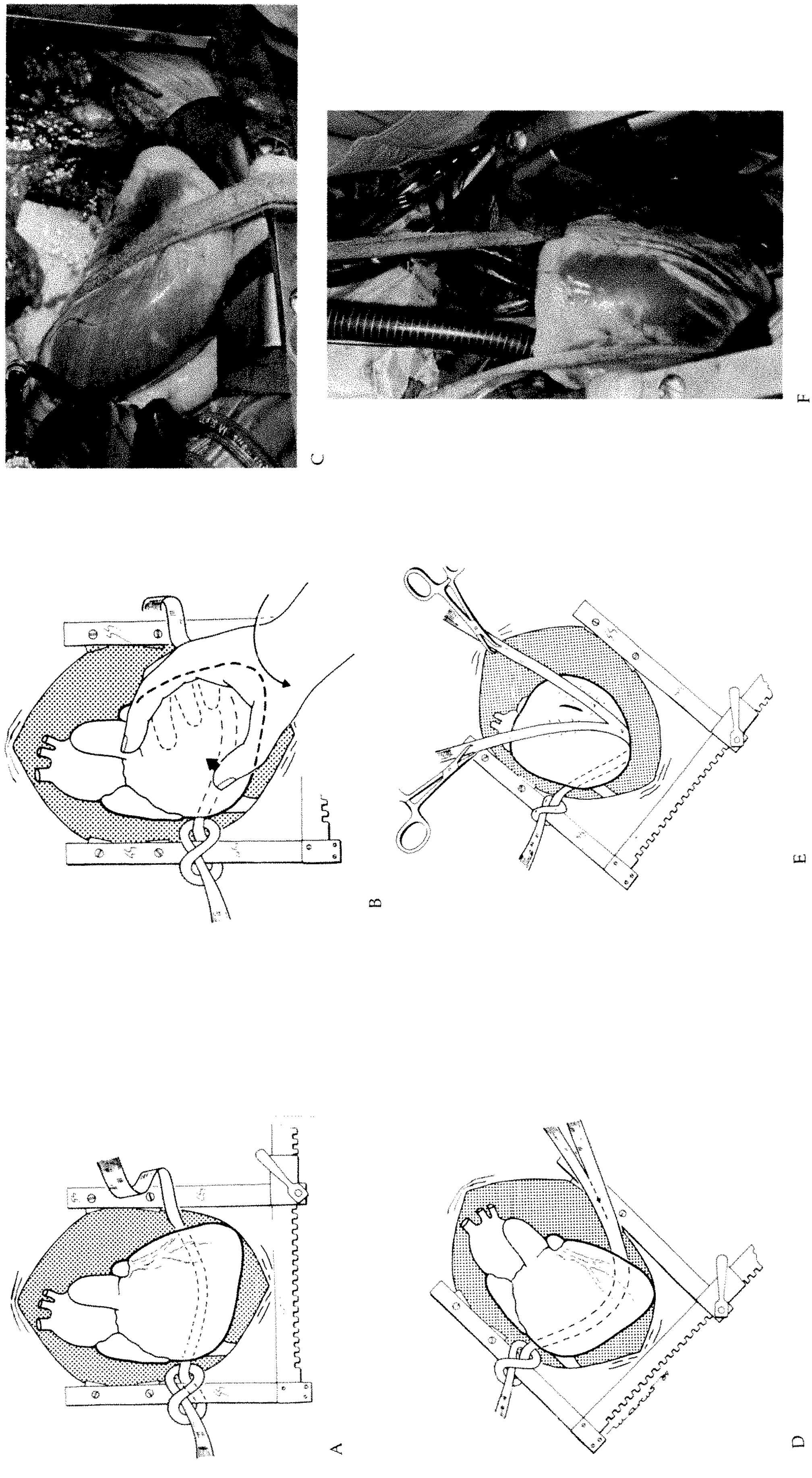

Fig 1. (A) Fixation of sling to chest retractor and passage through lateral wall of oblique sinus. (B) Positioning the right hand to give heart counterclockwise roll. (C) Exposure of obtuse marginal artery. (D) How to bifurcate the sling. (E) Bifurcated sling secured with towel clips positions convexly the distal marginal territory and makes the arteriotomy pout, enabling easy anastomosis. (F) Bifurcate sling retraction for distal marginal anastomosis.

Permanent Pacemaker Implantation: Retropectoral Position and Lead Insertion Through the Subclavian Vein

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A technique is described for surgical implantation of pacemaker leads using the subclavian vein and placement of the pacemaker posterior to the pectoralis major muscle.

(Ann Thorac Surg 1990;49:835–6)

Frequent complications of pacemaker placement in the subcutaneous position in elderly and emaciated patients are erosion of the skin and "twiddling" of the pacemaker [1]. To avoid these complications, we have used a technique of retropectoral placement of the pacemaker and cannulation of the subclavian vein for introduction of the permanent pacemaker leads.

Technique

A transverse skin incision approximately 6 to 7 cm in length is made 2 cm below the lateral half of the right clavicle with the patient under local anesthesia (Fig 1). The subcutaneous tissue is divided in the direction of its transverse fibers, which insert on the clavicle, exposing the pectoralis major muscle, which is divided exposing the subclavian vein. Two pursestring sutures of 4-0 vascular suture material are placed in the anterior wall of the subclavian vein. Snares are attached to these sutures and are used to control hemostasis during placement and positioning of the endocardial leads. A guidewire is then introduced into the subclavian vein and advanced into the superior vena cava. A disposable plastic sheath-dilator is passed over the guidewire, which is removed (Fig 2). The permanent electrodes are passed into the heart under radiological control and the plastic sheath is peeled off. Hemostasis at the exit site of the subclavian vein is controlled by use of the pursestring sutures and snares during positioning in the right ventricle and measurement of thresholds. Once position of the leads is satisfactory and stable, the pursestrings are tied and the ends of the sutures are tied around the leads to secure them in place. Using blunt dissection the posterior surface of the pectoralis major muscle is lifted off the chest wall and pectoralis minor muscle (Fig 3). This does not usually result in any bleeding. The pacemaker is attached to the leads and placed in the retropectoral pocket. Hemovac drains are reserved for patients under anticoagulation. The cut edges of the pectoralis major muscle are sutured together. The subcutaneous tissue and skin are closed in separate layers.

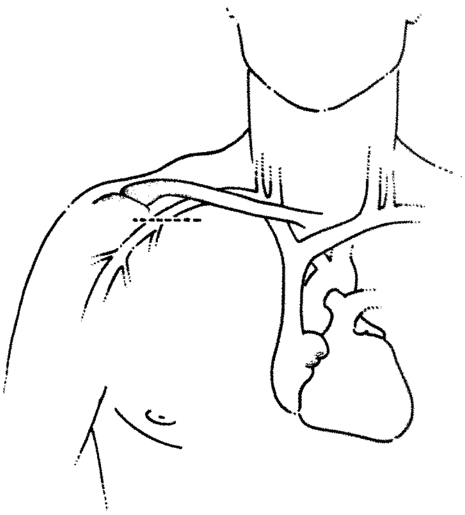

Fig 1. Site of the incision.

Comment

This technique has been used successfully between August 1975 and June 1989 in more than 70 patients. The

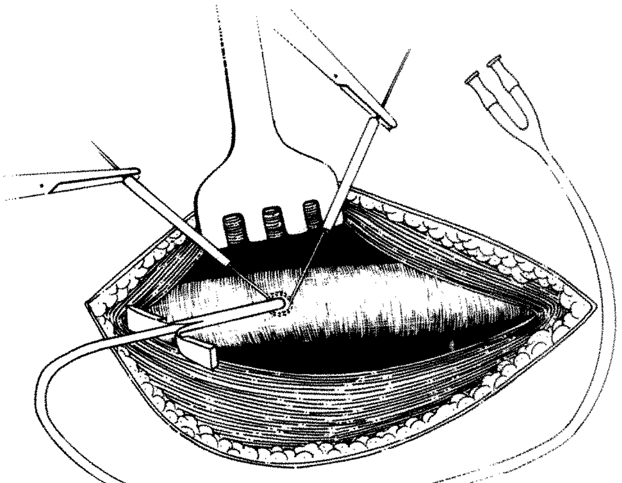

Fig 2. Exposed subclavian vein showing pursestring sutures and introducer with lead.

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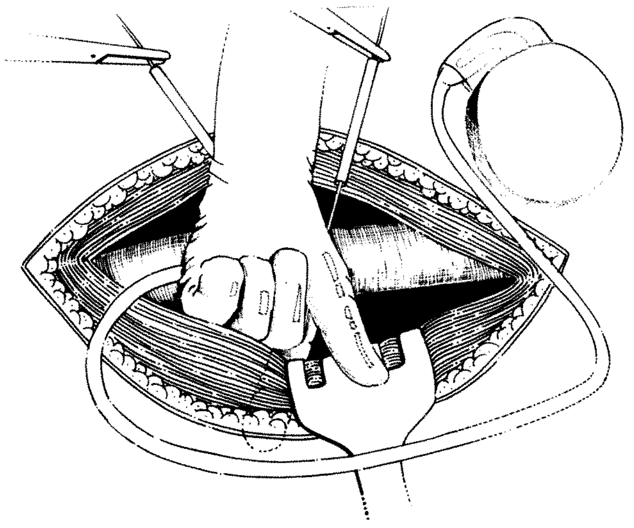

Fig 3. Fashioning the retropectoral pocket by blunt dissection.

only complication observed was one hematoma in the pocket 2 weeks after implantation in a patient under anticoagulation. For this reason, we recommend the use of a Hemovac drain in patients under anticoagulation therapy. The advantages of the technique are minimal blood loss, ease of introduction, an almost imperceptible position of the pacemaker, and impossibility of "twiddling"; no case of erosion has been noted as the pacemaker is protected by the pectoralis muscle in addition to the subcutaneous tissue.

An added advantage is that in young women the incision may be made lateral to the edge of the pectoralis major muscle for aesthetic reasons. Replacement of the pacemaker pack has not proved difficult as the same fibrous capsule forms around the pacemaker as it does in the subcutaneous position. Finally, there has never been a problem with inadequate vein size as may occur with the cephalic vein [2] or thrombosis of the subclavian vein. This technique uses elements of subcutaneous introduction but without the complication of pneumothorax [3] and applies aspects of vascular surgery that will appeal to the surgeon called on to implant pacemakers.

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Surgery for Ventricular Arrhythmia

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Years of frustration of cardiac surgeons attempting to control intractable ventricular arrhythmia finally ended when the team of Harken, Josephson, and Horowitz performed electrophysiologically directed left ventricular endocardial resection and reported their early results 10 years ago. The scientific background for this breakthrough is reviewed and some subsequent modifications of their procedure are described. The current status of arrhythmia surgery since the advent of the automatic internal cardioverter-defibrillator is described: the two methodologies are complementary, not competitive.

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Surgical Endocardial Resection for the Treatment of Malignant Ventricular Tachycardia

ALDEN H. HARKEN, M.D., MARK E. JOSEPHSON, M.D., LEONARD N. HOROWITZ, M.D.

A decade has now passed since the classic contribution of Harken, Josephson, and Horowitz to the surgical management of patients with intractable ventricular tachycardia unresponsive to medical management. This landmark contribution and the publication that described it ushered in the current era in which "surgical therapy of refractory ventricular tachyarrhythmias is now rational, recommended, and rewarding" [1].

If one of the hallmarks of a "classic" paper is rapid acceptance of a new method or technique, then this paper well qualifies: the method was quickly adopted in many centers throughout the US and the world. The necessity for this contribution arose from long frustration of surgeons attempting to cure patients with intractable ventricular arrhythmia, most often a complication of ischemic heart disease, by various surgical approaches such as sympathectomy, ventricular aneurysmectomy, intraaortic balloon pumping, and coronary revascularization. It had been widely felt that coronary revascularization would solve the problem by improving the blood supply to the border zone of infarction, and a number of publications attested to this "fact." But this optimism was short lived, except in that group of patients with primary ventricular fibrillation due to ischemia, and usually with a normal ventricle.

The basis for a rational surgical approach to ventricular arrythmia was long in coming, dating from the work of Sir Thomas Lewis, reported in 1915 [2], and from the elegant work of Durrer and associates [3] describing in 1970 the normal activation sequence in the isolated human heart. Wellens and co-workers [4] provided data suggesting that the reentry phenomena was the responsible mechanism for ventricular tachycardia in humans. About the same time, Fontaine and associates [5] followed by Spurrel and colleagues [6] and Wittig and Boineau [7] performed intraoperative epicardial and transmural mapping to guide limited transmural incisions or excisions in an effort to interrupt reentry pathways and cure the arrythmia. The latter paper, presented at the 1975 meeting of The Society of Thoracic Surgeons and published in this journal, stimulated our group to apply this methodology, which gave encouraging but still frustrating results [8].

It was in this setting, then, that Harken, Josephson, and Horowitz took the bold step of mapping the endocardial surface of the open, beating, normothermic heart during induced ventricular tachycardia. The findings, usually confirmatory of preoperative endocardial catheter mapping, were used to direct excision of endocardial scar in the area of earliest activation of the tachycardia, invariably in the sharply demarcated border zone between scar and normal myocardium. Their experience in the first dozen such cases, reported by Alden Harken at the 1979 meeting of the American Surgical Association, caught widespread attention even before publication in the October issue of the *Annals of Surgery* [1], and other groups began to follow their lead. As befits a classic, the article is a model of brevity and clarity, only four pages long, more than one of which is taken by illustrations (Fig 1).

As is often the case in medical breakthroughs, an intuitive approach to solution of a problem may be successful: laboratory data to provide a sound physiological basis may follow. In this case it was more than intuitive, backed by a solid background in clinical electrophysiological studies including endocardial mapping, but less than totally scientific. Their group, following initial clinical success, created a canine infarct model and were able to demonstrate subendocardial origin of tachyarrhythmias with variable, often distant, breakthrough to epicardium [9].

From the beginning of our effort in applying this new technique, and largely at the behest of cardiologist Richard Kehoe, we decided to make a departure from the Philadelphia technique and remove all visible endocardial scar while taking into full consideration the findings of intraoperative mapping. The reasons were several: approximately 25% of patients cannot be mapped at operation for a variety of reasons; many patients have more than one tachycardia morphology, as many as five in our

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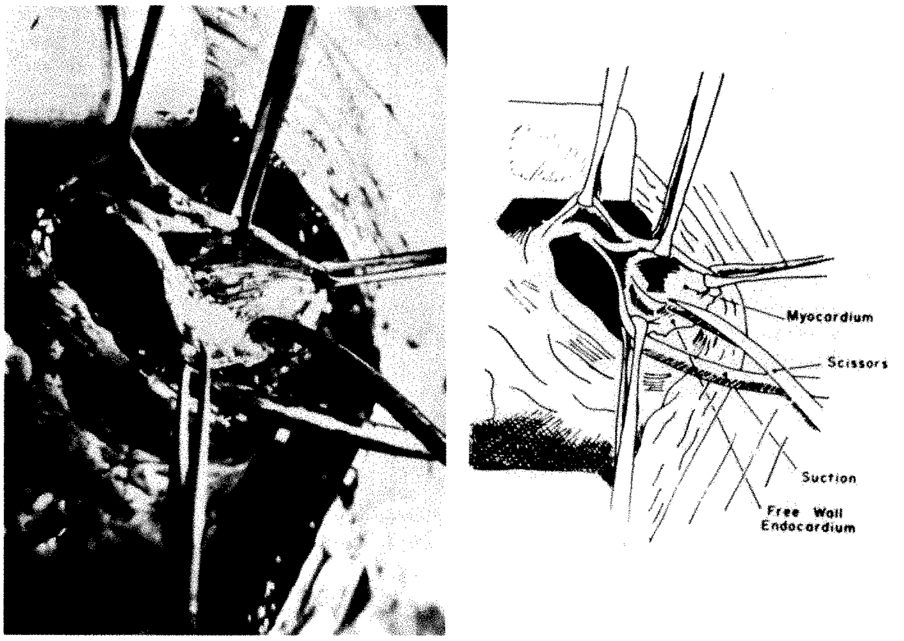

Fig 1. Operative photograph revealing a left ventricular aneurysmectomy. The tachycardia originated from the endocardial surface of the left ventricular free wall. The free wall endocardium is excised for 2–3 cm beyond the limits of the aneurysmectomy. (Reprinted from [1] by permission of J.B. Lippincott Co.)

experience, and different morphologies usually arise from distinctly different areas; arrhythmias may develop many years, up to 30 in our experience, after infarction, so it did not seem logical to leave abundant scar behind identical to that in the area of earliest activation to serve as a matrix for future arrhythmia, provided it was safe to resect [10]. Over the long term it appeared that this approach led to somewhat improved results as defined by noninducibility and a clinical course free of arrhythmia, with most patients off antiarrhythmic medication [11, 12].

The following year Harken's group published another important paper [13] which compared the results of aneurysmectomy alone for ventricular arrhythmia with those of aneurysmectomy combined with an electrophysiologically directed endocardial resection. This paper, presented at the 1980 meeting of the American Association for Thoracic Surgery was introduced and discussed by David Sabiston. He noted the special relationship between the sponsor of the paper, Dwight E. Harken, and the presenter, his son Alden. He remarked to the effect that the acorn had not fallen far from the tree in terms of pioneering efforts in cardiac surgery, both having made their signal contributions at a similarly youthful age. The marked success of the directed aneurysm resection (7% operative mortality and 10% inducible ventricular tachycardia) contrasted sharply with the poor results of nondirected resection (42% operative mortality and 26% late mortality, mostly arrhythmic). These results are in close agreement with our earlier nondirected aneurysmectomy series of 15 patients (60% early and late mortality, mostly arrhythmic) [8].

Electrophysiologic surgery of the heart by definition requires a team approach, the nature of which is perhaps unique in all of surgery. The interdependence of cardiologist-electrophysiologists and surgical teams is required to correlate anatomy with electrical events to localize the area(s) of early activation during ventricular tachycardia. An essential ingredient is the patience that each group must exercise with the other. I recall with amusement on our visit to Philadelphia, the happy hubris with which Josephson and Horowitz directed the mapping efforts, the extreme patience of Alden Harken, and his colleagues' hasty exit after completion of the exercise, tripping over and disconnecting the power supply to the heart-lung machine on their way out of the room. Their association, extremely productive, lasted a number of years, to be dissolved by the peregrinations so common in academia. At Northwestern, my association with Richard Kehoe and Jerod Loeb, the latter also in charge of our laboratory effort, was most congenial and rewarding. Another ingredient that is essential for such continuing innovation in surgery is an atmosphere conducive to clinical investigation, an academic environment in which innovation is encouraged. In generous measure, this was provided by Department Chairmen L. Henry Edmunds at the University of Pennsylvania and Lawrence L. Michaelis at Northwestern. Their important roles in contributing to these advances should also be recognized.

Constant flux in methodology being one of the few constants in surgery, the automatic internal cardioverter-defibrillator has had an enormous impact on the field of arrhythmia surgery. Long in development, often in adverse circumstances and without government support, Mirowski and associates [14] have provided a remarkable device that can detect ventricular tachycardia and fibrillation and terminate either with direct current countershock, of increasing energy if need be. Early on, many of us felt that patients receiving the automatic internal cardioverter-defibrillator would be denied surgical cure in favor of an expensive, unreliable device, trading one disease for another. However, with time and experience, it has become apparent that the automatic internal cardioverter-defibrillator and ablative methodologies are really complementary. As surgeons, we are pleased to have an operation with low mortality (2% to 3%) for our high-risk cases, and for those that clearly are inoperable for an ablative procedure. On the other hand, there are still many patients who need endocardial resection, cryolesions, or laser ablation of endocardium: those relatively young, with or without critical coronary disease, with discrete aneurysm or other clear anatomical target, with inducible malignant arrhythmia, and who are otherwise good operative risks. Their reward is a high likelihood of cure, without the need for antiarrhythmics; those having automatic internal cardioverter-defibrillator implantation virtually all need supplemental drugs of varying toxicity and periodic expensive generator replacement, and many have been subject to component failure with inappropriate sensing and discharge. However, most of these problems are on the way to solution because of the intensity of research and development for a new generation of devices that will have the ability to act as pacemakers, to terminate tachycardias by extrastimulus, as well as to countershock. They will come in smaller packages, and thoracotomy will no longer be necessary. So for that group of patients the thoracic surgeons will play a diminishing role in what will become a catheterization laboratory procedure. But the day when ablative surgery will no longer be necessary or desirable is not yet in sight.

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Lung and Heart-Lung Transplantation

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Unilateral lung transplantation has become a successful method for the treatment of end-stage pulmonary disease, whereas double-lung transplantation has provided benefit to patients with nonfibrotic lung disease such as emphysema and cystic fibrosis. In the past 5 years, 16 single-lung and 13 double-lung transplantations have been performed by the Toronto Lung Transplant Group in patients with end-stage lung disease. Seven perioperative and two late deaths have been recorded so far. Since the introduction of heart-lung transplantation at Stanford in 1981 and at Pittsburgh in 1982 for the treatment of Eisenmenger's syndrome and terminal pulmonary vascular disease, more than 350 combined heart-lung transplantations have been carried out throughout the world. Presently, the 2-year actuarial survival is about 62%. The long-term results have not yet reached the same level of success as those of cardiac transplantation alone. Although several factors have played a role in this difference, a prominent cause has been the lack of a reliable and simple method for pulmonary protection against prolonged ischemia. Most of the techniques proposed against ischemia can be classified as normothermic or static hypothermic cardiopulmonary preservation. The use of the normothermic method has not always been successful. For this reason, interest has now been directed toward the potential for hypothermic preservation of the heart-lung bloc and the use of free-radical scavenger therapy in the reduction of reperfusion injury.

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For more than three decades, the potential value of lung and heart-lung transplantation in the treatment of patients with end-stage lung disease has stimulated a large number of laboratory and clinical studies [1].

Lung Transplantation

In 1950, Matras [2] reported a major technical advance in single-lung transplantation in a canine model. A similar technique was independently described by Neptune and his group shortly thereafter [3]. In 1951, Juvenelle and associates [4] performed the first successful long-term reimplantation of the entire lung in a dog. In 1952, Hardin, Kittle, and Schafer [5] published their preliminary observations on canine left lung allotransplantation, after which numerous studies of reimplanted and allotransplanted lungs were reported [6].

The first human lung transplantation was performed by Hardy and associates [7] in 1963. Although the patient died of renal insufficiency after 18 days, the lung transplant functioned well and without rejection. In 1964, Meshalkin and associates [8] completed a series of lung reimplantation in patients with asthma. Although the condition of some of the patients seemed improved, these investigators gave up the procedure. However, their studies demonstrated that unilateral or staged bilateral reimplantation of the lung in humans could be successfully accomplished.

During the 20-year period that followed Hardy's original attempt at human lung transplantation, approximately 40 such procedures were performed in patients with end-stage pulmonary disease [9]. The results were disappointing: the mean survival was only 8.5 days; 2 patients survived for 6 and 10 months [10]. The high mortality was related to pulmonary infection, rejection of the transplant, and complications at the bronchial anastomosis. These complications included dehiscence, mucosal necrosis, infection, hemorrhage, and late bronchostenosis. Ischemia of the donor bronchus subsequent to division of the bronchial arterial supply, the rejection response that takes place at the anastomotic site, and the toxic effects of immunosuppressive drugs to prevent allograft rejection may all play a role in the pathogenesis of these complications [1].

Bronchial Anastomotic Healing

IMMUNOSUPPRESSIVE THERAPY. The adverse effects of steroid therapy on wound healing are well recognized. Many patients who died of bronchial anastomotic complications after lung transplantation had been on a steroid-based immunosuppressive regimen [9]. Lima and co-workers [11] have shown that the combination of steroids and azathioprine significantly reduces the breaking strength of both bronchial anastomoses and skin incisions after lung transplantation in dogs. The combined action of these immunosuppressive drugs interferes with the healing process by altering collagen deposition at the bronchial anastomotic sites and skin incisions.

Cyclosporine A, a powerful new immunosuppressive agent, has been used with much success in a variety of animal [12] and human organ transplants [13]. Laboratory studies have demonstrated that cyclosporine, unlike steroids, has no harmful effect on bronchial healing [14]. These results have made cyclosporine particularly suitable for immunosuppression in clinical lung transplants [9, 10], in which bronchial healing and pulmonary infection have been a serious problem. Cyclosporine is a cyclic

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polypeptide that exerts selective and reversible inhibition of stimulated T-lymphocyte proliferation by blocking the production of interleukin-2 [12, 13]. Variability of cyclosporine pharmacokinetics between and within drug recipients has created some confusion regarding the ideal dose regimen that will produce a consistent immunosuppression without causing adverse reactions [15]. These reactions include nephrotoxicosis, hepatotoxicosis, neuropathy, gingival hypertrophy, hirsutism, and endothelial cell injury. When detected early, most of these complications are reversible with drug withdrawal or dose reduction. Therefore, monitoring of blood cyclosporine levels before daily administration of the drug is necessary in the postoperative management of transplant patients [15].

REVASCULARIZATION TECHNIQUES. Many methods have been proposed to overcome bronchial ischemia and to improve bronchial healing after lung transplantation [1]; however, some of them add to the difficulty of the transplant operation and prolong the operating time. We have previously demonstrated [16, 17] in a canine model that the application of a pedicled intercostal muscle graft or peritracheal membranous adipose tissue flap on the bronchial anastomosis improved bronchial healing by restoring a vascular flow across the suture line seven days after lung autotransplantation. An omental pedicle flap has been used clinically by the Toronto Lung Transplant Group to revascularize rapidly the bronchial anastomosis [18]. In the past 5 years, 16 unilateral lung transplantations have been performed by this group [19] in patients with end-stage lung disease. No patient died of bronchial anastomotic complications. Good bronchial healing was observed in the majority of patients. Four in-hospital deaths and two late deaths have been recorded so far.

Conditions for Lung Transplantation

Unilateral lung transplantation is a rapidly developing procedure with demonstrated therapeutic efficacy and great potential for improved survival in select patients with end-stage pulmonary fibrosis [18, 19]. However, many patients with end-stage pulmonary disease are not suitable for unilateral lung transplantation. It is believed that in individuals with bilateral pulmonary sepsis such as cystic fibrosis, the remaining native lung would serve as a potential source of chronic infection in the immunosuppressive recipient after transplantation. There has been also some concern that, in patients with emphysematous lung disease, unilateral transplantation may give rise to excessive inflation of the native lung with a shift of the mediastinum toward the transplanted lung [18], even though unilateral transplantation has been shown to improve respiratory function in 2 of these patients [20]. Of interest is a recent report from France confirming the beneficial effect of this intervention for some individuals with emphysema [21].

Combined heart-lung transplantation has been used in a small number of patients with end-stage lung disease. However, it was soon recognized that many candidates who were considered for transplantation had "adequate or recoverable" right ventricular function. For this reason, the Toronto Lung Transplant Group adopted a logical approach that consisted in treating such patients by replacement of both lungs without removal of the native heart [18]. Hence, from November 1986 to September 1988, 13 double-lung transplantations were performed by this group. Three of the 13 patients died of tracheal complications, and serious impairment of airway healing developed in 7 others [19]. Indeed, ischemia of the donor trachea and proximal bronchus continues to be a serious problem with the current double-lung transplantation procedure. It appears that the omental pedicle alone is not sufficient to provide adequate blood supply to the proximal donor airway. Other approaches are needed to clear up this problem in the future. Recently, Cavarocchi and Badellino [22] described in their heart-lung transplantation procedure a technique that consists of preserving the recipient's pulmonary artery for "later use as a pedicle flap to the tracheal anastomosis." Although postoperative complications were observed in their 3 heart-lung transplant patients, none of these complications included impairment of airway healing.

During the past few years, a substantial subset of patients with end-stage lung disease but with satisfactory cardiac function have served as donors of cardiac allografts before undergoing heart-lung transplantation. This "domino-donor" operation has been performed in 12 patients in the United States and in more than 30 patients in England [23]. One potential advantage of the procedure is that it avoids wasting a usable heart. On the other hand, Cooper and associates [24] believe that en bloc double-lung transplantation is a "more reasonable approach" because it excludes many of the complications due to the cardiac portion of the transplant such as chronic and acute rejection as well as proliferative coronary vascular disease. It also eliminates the unnecessary removal of the heart from a patient who does not need cardiac replacement. Indeed, combined en bloc double-lung transplantation, as with unilateral lung transplantation, allows the use of the lungs from a donor after removal of the heart for transplantation, and therefore considerably increases the number of donor organs for patients with end-stage pulmonary disease.

One important problem in the field of lung transplantation is the diagnosis of pulmonary rejection. Presently, the diagnosis is based on clinical findings such as lung infiltrates, chest roentgenogram opacities, a decline in arterial oxygenation, leukocytopenia, and mild fever. A positive response to a pulse dose of steroids is considered a confirmation of rejection. Transbronchial biopsy, bronchoalveolar lavage, and examination of peripheral blood for lymphocyte subsets are now being evaluated in many institutions.

Heart-Lung Transplantation

With the introduction of cyclosporine in the late 1970s, it was found that lung grafts in animals healed more readily when the recipients were treated with the new immunosuppressant. Nevertheless, because of inadequate revascularization of the donor bronchus, dehiscence was still frequent [1]. To prevent this problem, one alternative approach was proposed: the transplantation en bloc of the heart and lungs.

In 1981, heart-lung transplantation was introduced at Stanford University as a therapeutic possibility for patients with terminal pulmonary vascular disease [25]. The decision to carry out this program was based on four factors: (1) the wide experience the Stanford group had gained in cardiac transplantation, (2) the availability of cyclosporine, (3) the disappointing worldwide results of clinical lung transplantation, and (4) the ability to achieve long-term survival after joint heart and lung transplantation in primates. Combined heart-lung transplantation was first performed in humans in 1968 by Cooley and associates [26], who based their operative procedure on laboratory investigations described by Webb and Howard in 1957 [27] and Lower and colleagues in 1961 [28]. From 1969 to 1971, two more such procedures were performed in humans [29, 30]. There were no long-term survivors from these early clinical efforts. Since 1981, more than 350 combined heart-lung transplantations have been performed throughout the world. A few cases of emphysema and cystic fibrosis were treated with heart-lung transplantation [31], but primary pulmonary hypertension and Eisenmenger's syndrome remained the predominant indications for the procedure [32].

In the early experience with heart-lung transplantation, 40% to 50% of patients died in the first year, many of them in the perioperative period. Improvement in donor and recipient selection, operative technique, and immunosuppressive therapy along with continuous observation of infection and rejection should lead to better results. Of 219 combined heart-lung transplantations carried out worldwide from 1986 to 1988, the 2-month actuarial survival is more than 70% but drops to 62% at 2 years [32]. The long-term functional results of combined heart-lung transplantation have been frustrating as a result of the substantial incidence of obliterative bronchiolitis.

Indeed, heart-lung transplantation therapy is difficult and complicated, and the present intermediate and long-term results have not yet reached the same level of success as those of cardiac transplantation alone [31, 32]. Although several factors such as selection of candidates and donors, technical execution of the operative procedure, and prevention of postoperative infection have played a role in this difference, a prominent cause has been the inability to provide adequate extended pulmonary preservation [31].

Ex-Vivo Preservation of the Heart-Lung Bloc

Unlike cardiac preservation techniques that now allow prolonged ischemic times sufficient for distant procurement, the lack of a reliable and simple method for pulmonary preservation has for some time prevented distant procurement of the heart-lung bloc, necessitating the transfer of brain-dead, heart-beating donors to the transplant institution. This, together with the strict donor selection criteria, has resulted in a serious shortage of suitable donors [33].

Most of the techniques proposed for the distant procurement of combined heart-lung grafts can be classified as normothermic or static hypothermic cardiopulmonary preservation.

Normothermic Cardiopulmonary Preservation

Normothermic cardiopulmonary preservation involves the use of the autoperfused working heart-lung preparation. This method, first described by Martin and later by Starling, has been investigated by many researchers as a preservation modality for heart-lung transplantation [34]. Robicsek and associates [35], the first group to use this technique to maintain prolonged ex-vivo heart preservation, recently achieved 18 to 22 hours of myocardial and eight to ten hours of pulmonary preservation by adding metabolic substrate to their canine normothermic autoperfused working heart-lung preparation. Ladowski and associates [34] evaluated the autoperfused working heart-lung technique as a possible means to transport human organs over a long distance. Their method of preservation was reliable up to four hours only. The Johns Hopkins Hospital Group [36] examined this method in their surgical laboratory and showed that the addition of isoproterenol and metabolic substrate to the autoperfused circuit could extend cardiopulmonary preservation up to 18 hours. Hardesty and Griffith at Pittsburgh [37] reported successful clinical use of the autoperfused working heart-lung method for distant procurement of the heart-lung bloc with extracorporeal preservation times exceeding four hours. Miyamoto and colleagues [38] studied physiological changes in a modified model of autoperfused canine heart-lung preservation in which the preload was kept constant by interposing a compliance chamber in the arterial line. Lung function remained quite stable for up to five hours of preservation, but then it began to deteriorate. Complement activation, trapping of white blood cells, and platelet aggregation contributed to the deterioration.

Although the use of a normothermic autoperfused working heart-lung preparation has resulted in a few successful distant heart-lung preservations and subsequent transplantations, this method remains technically demanding and its clinical application is further complicated by several pitfalls [31, 34, 37, 38]. For this reason, interest has now been directed toward the potential for hypothermic protection of the heart-lung bloc.

Static Hypothermic Cardiopulmonary Preservation

This technique includes single-flush perfusion by way of the pulmonary artery and core cooling of the donor on extracorporeal bypass followed by cold storage.

SINGLE-FLUSH PERFUSION OF THE PULMONARY ARTERY. Perfusates that have been investigated for flushing the pulmonary vasculature may be characterized as of the intracellular type or extracellular type with and without colloid [39]. Extracellular type of solutions such as Ringer's lactate, isotonic saline solution, plasma, and dextran have been shown to be inadequate for lung preservation [39]. Two types of intracellular solutions containing low sodium and high potassium concentrations and large amounts of phosphate bases have been used successfully in lung preservation for both flushing and storage: the Collins and the Sacks solutions [38].

Since 1981, a modified Collins solution (with 12 mEq/L of magnesium sulfate) has been used successfully at Stanford University for flush perfusion of the pulmonary artery at a low flow rate during on-site harvesting of the heart-lung bloc [40]. Additional topical cooling was achieved with cold Physiosol extracellular electrolyte solution (Abbott Laboratories, Quebec, Canada) at 4°C. In this setting of brief ischemia, the lung remained adequately preserved. However, in experimental studies with extended ischemic times, the results have been inconsistent. Feeley and colleagues [41] evaluated in a canine model the effect of hypothermic flush perfusion and five-hour cold storage on heart-lung preservation with crystalloid and Collins solutions. Each preservation technique was associated with large increases in extravascular lung water after implantation. These increases were consistent with pulmonary edema and insufficient ischemic preservation. Starkey and colleagues [42] reported that pulmonary artery flush with large volumes of intracellular fluids not only provided a more rapid and complete cooling of the graft but also elicited a pronounced pulmonary vasoconstriction at the beginning of flushing. Based on this information the Stanford group [40] used prostaglandin E_1 to enhance vasodilatation of the pulmonary bed during cold flush perfusion of the pulmonary artery. Prostaglandin E_1 is a powerful vasodilator capable of reducing the activity of some blood cells, particularly neutrophils and platelets. In addition, it can selectively influence the T and B lymphocytes [40]. In their recent studies in primates, Harjula and his associates [43] demonstrated that the preservation of the heart-lung bloc against an ischemia of six hours could be achieved with success if the donor was treated with prostaglandin E_1 immediately before pulmonary artery perfusion. Hakim and associates at Papworth [33] reported successful human heart-lung transplantation after distant organ procurement with three-hour cardiopulmonary preservation. Based on the principle of a single cold pulmonary flush, their method uses the infusion of prostacyclin before and in addition to the flushing solution.

CORE-COOLING OF THE DONOR ON CARDIOPULMONARY BYPASS. Cardiopulmonary bypass and deep hypothermia to cool the donor organs without flush perfusion of the pulmonary artery has provided short-term preservation of the heart and lung [44]. The procedure involves the harvest of organs together with the dissection of the heart-lung bloc while the latter is being perfused by extracorporeal circulation. This method, which has also been used for distant procurement by the Johns Hopkins [23] and Harefield groups [45], has provided protection of the heart-lung bloc against ischemic times not exceeding four hours.

Wahlers and co-workers [46] compared cardiopulmonary bypass with deep hypothermia with flush perfusion of the lungs using Eurocollins solution in dogs undergoing heart-lung transplantation. After six hours of preservation, only the pulmonary vascular resistance was significantly lower in the group of organs preserved by extracorporeal circulation. This would imply that lung preservation might be superior using that method. Bando and associates [47] recently reported that retrograde heart perfusion with oxygenated intracellular solution containing perfluorochemicals, lung immersion in extracellular solution, the administration of liposomal superoxide dismutase, and no flush core cooling could provide adequate cardiopulmonary preservation for 12 hours. In a recent experimental study in which current organ preservation methods for heart-lung transplantation were evaluated, Fraser and associates [48] concluded that donor core cooling provides better graft protection and therefore may be the method of choice for distant organ procurement.

Reperfusion Injury

Evidence is now accumulating that oxygen-derived free radicals are implicated in a large variety of pathological conditions, particularly in the pathogenesis of myocardial and lung injury occurring with ischemia and reperfusion [47]. Recent studies suggest that hypothermic perfusion of the isolated organ with an oxygenated solution may increase the rate of oxygen free-radical production [49]. Moreover, these studies have indicated that the combination of two specific enzymes and antioxidants, superoxide dismutase and the hydroperoxidase catalase, can provide significant myocardial protection when added to the cardioplegic solution. Superoxide dismutase, alone or in combination with catalase, has also been shown to play an important role in the prevention of lung reperfusion injury after periods of ischemia [50].

Distant procurement with adequate lung preservation is one of the important determinants of early and long-term results of heart and lung transplantation. Ischemic reperfusion injury, which contributes to the problem of lung preservation, is also a factor limiting the usefulness of distant organ procurement in heart-lung transplantation. Free radical scavenger therapy in the reduction of reperfusion injury may play a major role in the future of clinical lung and heart-lung transplantation.

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The Trachea

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CORRESPONDENCE

Crossovers in Coronary Artery Bypass Grafting Trials

To the Editor:

We read with interest the editorial by Fisher and colleagues entitled "Crossovers in coronary artery bypass grafting trials: desirable, undesirable, or both?" [1], which commented on our article "The effect of crossover on the statistical power of randomized trials" [2]. We believe that several of their points require comment.

First, they criticize our article for failing to consider the "definition of the relevant medical question," which they define as: "if we postpone CABG [coronary artery bypass grafting] until angina dictates operation and then allow operation, is survival adversely affected?" Unfortunately, this "relevant medical question" was *not* the question the Coronary Artery Surgery Study (CASS) was designed to answer. The Coronary Artery Surgery Study was "a randomized trial of coronary artery bypass surgery" [3] and was intended as a "comparison of the effects of randomized medical and surgical *treatment* of mildly symptomatic patients with coronary artery disease." [4] [emphasis added]. It sought to compare surgical versus nonsurgical therapy. It said nothing at all about comparing "delayed CABG" with "early CABG." Moreover, it lacked the structure, the sample size, and the statistical power to make meaningful comparisons of such a study question. Fisher and colleagues have inadvertently taken an inference drawn from the data and substituted it for the original experimental hypothesis, thus undermining the logic of the prospective study design.

Second, they claim that we did not use the best method to compute statistical power. In fact, there are only trivial differences between power calculated with our model versus their model in the CASS baseline article [4]. The powers calculated by the two models are virtually identical (see Appendix). In any event, our article stated that a simple binomial model was used for didactic purposes to demonstrate the profound effect of crossover on statistical power. We cautioned that appropriate calculations must be used when analyzing trial data by actuarial methods [2].

Third, they misinterpret our point when they claim that we suggested alternatives to the intention to treat principle, such as dropping crossovers from the study. Our paper makes it quite clear that we were rejecting such alternatives because of the bias they create. Our purpose was to show that crossover may create insurmountable problems in the analysis of such studies.

In addition, Fisher and colleagues mistakenly assert that we "assume that the mortality rate in the noncrossovers remaining in the medical group is the same as the mortality rate would be in the entire group of medically assigned patients if crossover were not allowed." We do not. In fact, we assumed a constant risk of death across patients merely to provide a point of departure for other, more realistic estimates [2]. Nowhere in the ensuing discussion does our analysis assume that the mortality rate in the noncrossovers, p_n, must remain constant when considering different crossover rates. Indeed, our model guarantees that p_n will vary, depending on the crossover rate, p_c, and the overall medical group rate, p_o. Thus, in the hypothetical example presented by Fisher and colleagues, the value of p_n simply decreases to p_o if crossover is not allowed. Because the mortality rate for the assigned medical group in the intention to treat analysis is, in reality, a mixture of rates for those undergoing operation and those remaining with medical treatment, exactly as observed, it is unclear to us why Fisher and colleagues think that use of our model "would yield a spuriously high estimate of mortality in the noncrossover group" [1].

Although the CASS baseline article [4] made some theoretical power calculations (which, incidentally, were as low as 0.32), CASS did not report the statistical power of the actual results [3]. The failure to do so makes it impossible to interpret the failure of observed differences to attain statistical significance and may lead to misinterpretation of the data. For example, the surgically assigned group had a lower mortality than the medically assigned group, but the difference was not statistically significant [3]. This finding was widely interpreted as showing no improvement with "surgery" (ie, surgical assignment) without considering that the failure to reach statistical significance may well have been due to the high probability of a type II error. Therefore, the conclusion that patients may "safely defer surgery until symptoms worsen" [3] was unwarranted in view of the low statistical power of the study [5].

The statement by Fisher and associates that "although many patients assigned to medical therapy later received CABG, the majority did not" may be misleading because it does not address the fact that the CASS crossover rates at 5 years were 26% overall and 38% in those with triple-vessel disease. Such high rates of noncompliance would vitiate almost any study, even though "the majority" received their assigned treatment.

Finally, in response to our statement that "not all randomized studies can guarantee results that are both valid and clinically applicable," Fisher and colleagues reply that "if randomized trials are abandoned, the issues would be decided based on purely observational data. . ." Certainly, we do not propose to abandon randomized studies; they are excellent tools for certain types of clinical questions. However, when nonnegligible crossover subverts the clinical intentions of the trial, such studies may lead to equivocal or frankly misleading conclusions. To fail to recognize this fact is to do a grave disservice both to our patients and to the authority of the randomized study design.

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Appendix

For example, with assumed rates of 2% of medically treated patients dying each year, 1% of surgically treated patients dying each year, and 5% of remaining medically treated patients

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transferred for surgical treatment each year, the CASS authors calculate a power of 0.754 for a trial with 390 patients per group using a one-tailed test [4]; with a two-tailed test, their power would be 0.645. Under these assumptions, the Markov model used in the CASS baseline article implies 5-year mortality rates of 9.2% for the medically assigned patients and 4.9% for the surgically assigned patients, and an overall 5-year crossover rate of 21.7% from medical to surgical treatment. Now consider our simple binomial mixture model [2], with p_1 = 4.9% for the surgical mortality rate and p_c = 21.7% for the crossover rate. Adopting p_n = 10.3% for the mortality rate among medical noncrossovers, so that the mixed rate in the medical group is p_o = 9.2% as required, we find a one-tailed power of 0.752 and a two-tailed power of 0.643, almost identical with the estimates obtained by the method of calculating power used in the CASS baseline paper [4].

Reply

To the Editor:

With all previous studies showing that coronary artery bypass grafting relieves angina pain more successfully than medical therapy, the CASS study always recognized that some of the medically treated patients would need to undergo operation owing to inadequate pain control on medical therapy. This is implicit in the appendix on statistical power [1], in which the medical survival is estimated including survival after crossing over to surgical therapy. The CASS manual of operations from the beginning had a section detailing allowable crossover; this was allowed after every effort had been made to achieve satisfactory angina control with medical therapy. Large amounts of crossover decrease statistical power, and for this reason, crossover was discouraged except when ethically necessary. Indeed, the criteria for changing assignment to therapy were discussed in the baseline paper [1]. Early in the study we did not anticipate the debate about such alternative phrases as "comparing medical and surgical treatment" and "comparing assignment to early medical and early surgical treatment."

Our statement that Weinstein and Levin "assume that the mortality rate in the noncrossovers remaining in the medical group is the same as the mortality rate would be in the entire group of medically assigned patients if crossovers were not allowed" is based on their formulas in the appendix [2]; this may or may not be a misinterpretation of their model. It seems unnatural to fix the medical survival (with crossovers included) and then to change the survival in noncrossovers depending on the crossover rate to get the fixed value as is suggested in their letter.

The letter mentions the CASS failure to present the statistical power of the results [3]. After the data are collected, we believe that confidence intervals are superior to statistical power; the paper would have been improved by presenting such intervals. However, for patients with good ejection fraction, with the 5-year survival being 95% in both groups, it is difficult to make a strong case for one strategy over the other.

We agree with much of what Weinstein and Levin have said. Crossovers do diminish statistical power for detecting differences. As mentioned in our editorial, one must address the relevant clinical question. Although CASS randomized more patients than any other randomized study in medical therapy and CABG for angina, we agree that the CASS study, and for that matter any study, would be strengthened by a larger sample size. Certainly in any study, the failure to find statistically significant treatment differences does not exclude any treatment effect whatsoever.

In response to Weinstein and Levin's last paragraph, we agree with these comments but we hope they are not applying them to CASS. Crossovers were anticipated as part of the assigned medical treatment; a mandated absence of crossovers would have no relevance to the current practice of cardiology. We do not believe that crossover subverted the intentions of CASS or that CASS has reached misleading conclusions.

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Cell Viability in Fresh, Refrigerated, and Cryopreserved Human Heart Valve Leaflets

To the Editor:

Viability may be defined as the ability of a cell or tissue to maintain itself and interact in a normal manner with its environment. Several studies have demonstrated heart valve leaflet fibroblast viability both during cold storage postmortem and after cryopreservation [1–8]. Clinical results indicate that viable cryopreserved allografts function better and longer than nonviable valves [9]. Furthermore, survival of donor fibroblasts in an allograft valve has been documented [9]. Therefore, it was most disconcerting to encounter the opinion in a recent editorial [10] that cryopreserved heart valves are definitely nonviable.

In our experience the leaflets of human heart valves are alive after up to 24 hours of ischemia, postmortem, and at that time approximately 80% of the fibroblasts exhibit tritiated-proline incorporation into collagen by autoradiography. This is an excellent method for demonstrating cell viability, because each cell that is actively synthesizing collagen can be observed in histological sections. After cryopreservation a mean of 70% of the fibroblasts are actively synthesizing collagen by this assay. The overall viability can then be obtained by dividing the experimental mean by the control mean (70 divided by 80), which gives a mean (± standard deviation) of 88.5% (± 12.27, n = 8) viable fibroblasts.

Viability can also be demonstrated by tritiated-glycine incorporation into proteins produced by cells, which include collagen. This is a routine assay for a cell biology laboratory. The leaflets are incubated in a solution containing tritiated glycine for 48 hours. Incorporation is assessed by scintillation counting of homogenized valve leaflets after protein precipitation and extensive washing to remove unincorporated radiolabel. Using this assay, the relative viability of nontransplantable human heart valve leaflets (with the appropriate donor consent) was assessed either immediately, after 1 and 2 weeks of refrigeration, or after cryopreservation. The results are expressed as a percentage of the fresh control values, which were obtained immediately. The three leaflets from each valve were bisected; one half of each leaflet was used as an immediate fresh control and the remaining half was used for one of the three experimental groups. There were no significant differences between 14 consecutive sets of cryopreserved valve leaflets (mean viability, 69%) and leaflets refrigerated for 1 week (mean viability, 70%), but both groups were significantly different ($p < 0.01$, Students t test) from the leaflets refrigerated for 2 weeks (mean viability, 13.2%). This experiment demonstrates that the majority of heart valve leaflet fibroblasts can survive cryopreservation or at least 1 week at refrigeration temperatures.

Such demonstrations of heart valve fibroblast viability, both postmortem and after cryopreservation, support previous reports in the literature. The fibroblasts of such heart valves have been shown to be alive by proliferation in vitro [3–5], metabolic activity resulting in pH changes [1], glucose utilization [1], general protein synthesis [2, 3, 5–7], and collagen synthesis [2, 5, 8]. Care must be taken to employ appropriate methods of tissue procurement, cryopreservation, and quality control [11, 12]. If this is done and the process is validated by proven viability assays, heart valve leaflets will be viable at transplantation. Then and only then can a cryopreserved allograft transplant series be compared with the results of O'Brien and colleagues in Brisbane [9].

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Reply

To the Editor:

The definition of *life* has been debated since time immemorial not only by biologists but also by philosophers and theologians. We have a good idea now as to what constitutes death, but the precise elements that make up life are not crystal clear. The same may be said of heart valvar viability. The proliferation of fibroblasts in tissue culture is probably not sufficient evidence for valvar viability. Endothelial and smooth muscle cells are also important constituents of the valvar apparatus. I think considerably more evidence is required before we can attribute the title of viability to cryopreserved human heart valves. This is not to say that Brockbank is mistaken in his interpretation of the experimental data. It is merely to imply that life has considerably more characteristics than the demonstration of 70% of fibroblasts actively synthesizing collagen.

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Porcine Versus Pericardial Bioprostheses

To the Editor:

We read with great interest Dr Pelletier's article "Porcine versus pericardial bioprostheses: a comparison of late results in 1,593 patients" [1]. This article is particularly useful in a period in which bioprostheses in general and pericardial valves in particular are in dispute. We completely agree with Dr Frater's [2] opinion that pericardial valves cannot be grouped together and that conclusions must be specific for each type of pericardial heart valve (PeHV). We had a similar experience [3]. For market reasons, we used four different types of PeHVs.

From January 1977 through June 1988, 200 patients received 242 PeHVs: Ionescu-Shiley, 124; Vascor, 56; Carpentier-Edwards 54; and Implamedic, 8. Patients' age ranged from 12 to 74 years (mean age, 48.2 ± 13.6 years). Follow-up was 955 patient-years and 1,194 valve-years, and was 95% complete. Actuarial freedom from valve failure was 61.9% ± 13.9% at 11 years for all PeHVs. In detail, it was 68% ± 14.8% for Ionescu-Shiley at 11 years, 53.2% ± 22.2% for Vascor at 7 years, 72.2% ± 21.5% for Implamedic at 5 years, and 100% ± 0% for Carpentier-Edwards at 4 years (Fig 1). Twenty-two PeHVs were explanted for malfunction: 14 for calcifications and 8 for tears. We observed calcifications and tears in the Ionescu-Shiley valve group; tears alone were indeed observed mainly in the Vascor and Implamedic valve groups.

It is interesting to note that if we compare PeHVs and our experience with porcine heart valves [4] we find significant differences only in the first years, but not at 10 and 11 years, probably because of the wide standard deviations of the PeHVs (Fig 2). At 11 years, freedom from valve failure for all porcine heart valves is 78.6% ± 3.7% [4]. But if we exclude Vascor and Implamedic valves, which gave us early unfavorable results, and

Fig 1. Actuarial freedom from valve failure for the four types of pericardial heart valves. (CE = Carpentier-Edwards; IM = Implamedic; IS = Ionescu-Shiley; VA = Vascor.) (Reprinted from [3] by permission of Casa Editrice Scientifica Internazionale Periodici.)

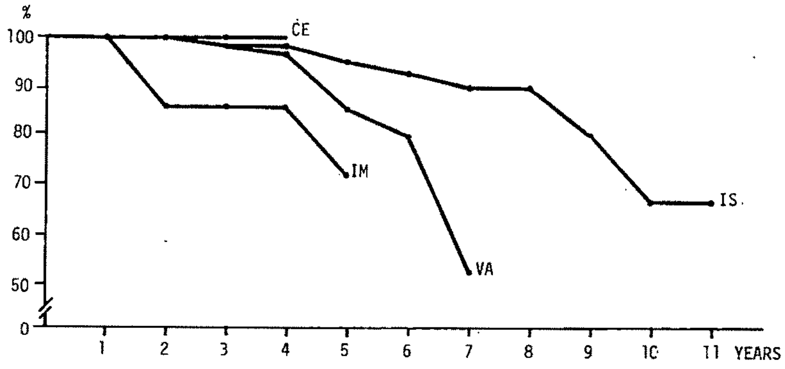


Fig 2. Actuarial freedom from valve failure for porcine heart valves (PoHVs) and pericardial heart valves (PeHVs). (Reprinted from [3] by permission of Casa Editrice Scientifica Internazionale Periodici.)

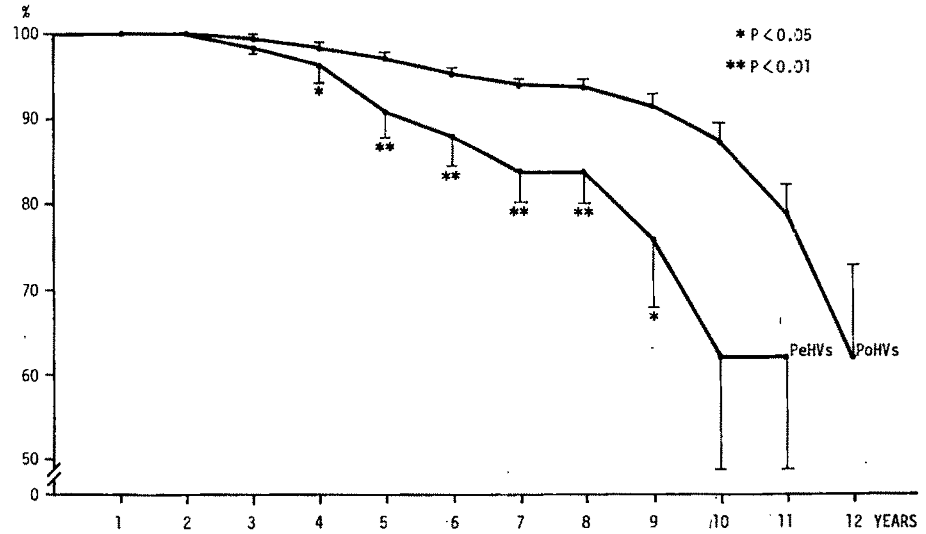
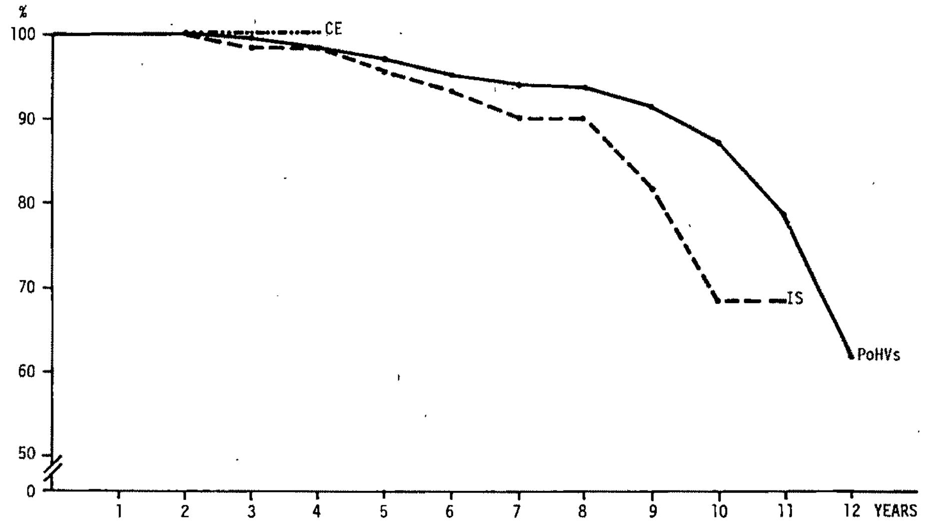


Fig 3. Actuarial freedom from valve failure for Carpentier-Edwards (CE), Ionescu-Shiley (IS), and porcine heart valves (PoHVs). (Reprinted from [3] by permission of Casa Editrice Scientifica Internazionale Periodici.)



compare only the Carpentier-Edwards and the Ionescu-Shiley valves against the porcine heart valves, we cannot find any significant difference (Fig 3). This underscores that PeHVs must be separately assessed and that there are thousands of patients, as Dr Gabbay said [5], with Ionescu-Shiley PeHVs that are still doing very well. Therefore, PeHVs have an acceptable performance and represent a valid choice as heart valve substitutes.

In particular, the Carpentier-Edwards valve appears to be a very good prosthesis with no instance of failure up to now. This substantiates Dr Pelletier's conclusion about the superior performance of the Carpentier-Edwards, but of course longer follow-up is mandatory to assess this valve definitely.

In conclusion, we appreciate Dr Pelletier's article, and we are convinced that in this critical time, in which many surgeons are discontinuing the use of bioprostheses in general and of pericardial valves in particular, a word of caution is mandatory before banishing these valves.

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Thoracoscopic Ablation of Blebs

To the Editor:

It is with a touch of envy and frustration that I read the nice report of Dr Wakabayashi [1], who successfully treated 9 of 10 patients with spontaneous pneumothorax using thoracoscopic electrocoagulation to ablate blebs.

Several years ago, I tried virtually the same procedure in 3 patients. Double-lumen endotracheal intubation and grasping bipolar forceps-coagulator were used. A Storz laparoscope was introduced through the chest tube site, with excellent visualization of apical blebs. No other skin incision was required. All patients were young and did not have other pulmonary pathology.

The first patient did well initially, but suffered recurrence 2 months later. The second patient continued to have air leak postoperatively. The possibility that some small blebs were missed during the procedure was entertained, so in the third patient, two surgeons inspected the pulmonary apex carefully and coagulated every conceivable leak and bleb site. Nevertheless, postoperative air leak persisted in this patient also. Transaxillary blebectomy cured all 3 patients. The pathological specimens showed interstitial hemorrhage and coagulation necrosis.

We gave up our trial of this procedure at that time with the conclusion that electrocautery coagulation did not provide reliable sealing for the air leak. This procedure has obvious advantages if it can be carried out with consistent success. I am awaiting with interest the experience of other surgeons.

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Reference

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Reply

To the Editor:

During 1989 alone I performed 33 thoracoscopies, both diagnostic and therapeutic, including 12 on patients with spontaneous pneumothorax. The experience gained by the routine use of thoracoscopy apparently plays an important role in achieving a high success rate of thoracoscopic procedures.

The success of thoracoscopic ablation of blebs or bullae depends on the visualization of the entire lungs and the ability of the surgeon to recognize the air leak. Although the distribution of blebs is usually confined to the apex it is not uncommon to find unexpected ones in the lower lobe. If the apex of the lung is adherent to the chest wall, it has to be freed because frequently the point of air leak is hidden behind it. If the point of air leak cannot be readily found, normal saline solution is poured into the pleural space through a suction probe while the lung is hand-ventilated. First the side of bubbling is determined, anterior or posterior. Then we concentrate on the side from which bubbles come to identify the hole. A ruptured bleb is usually small and cannot be seen as a hole but is always covered with fibrin materials. A ruptured emphysematous bulla is usually large enough to be identified as a hole.

Generally the air leak will stop within 24 to 48 hours after thoracoscopic treatment. If the air leak persists more than 48 hours, the patient should again undergo thoracoscopy without delay as it is likely that the lesion has been missed. The patient's acceptance of repeated thoracoscopy is excellent.

Out of 22 patients with spontaneous pneumothorax, I missed the air leaks in 2 patients, and these were successfully sealed by the repeated thoracoscopic thermocoagulation. The failure that Dr Chiu experienced appears to be due to failure to recognize the point of air leak. As for any other procedure, experience will improve our ability to identify the point of air leak and thus improve the overall result. So far, there has been no recurrence for up to 18 years. I would recommend that you electrocoagulate emphysematous bullae or blebs if they are encountered during routine thoracotomy to see how thermocoagulation works.

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Aprotinin and Open Heart Operations

To the Editor:

My colleagues and I read with interest the recent article by Alajmo and co-workers [1] regarding the hemostatic effects of high-dose aprotinin in open heart operations. As we are in the epicenter of the current acquired immunodeficiency syndrome epidemic, and are on occasion requested to perform open heart operations on patients positive for the human immunodeficiency virus [2], we suggest that use of aprotinin be considered in patients with human immunodeficiency virus–related thrombocytopenia.

A 34-year-old man who was a former intravenous drug abuser and positive for human immunodeficiency virus but had no evidence of acquired immunodeficiency syndrome was referred to us for surgical intervention. He was employed, and had been drug-free (on methadone maintenance) for 5 years. He had had *Streptococcus Sanguinis* endocarditis, possibly related to a dental procedure, and while free of active infection had a severely incompetent aortic valve with New York Heart Association class IV congestive heart failure, hepatic dysfunction, and human immunodeficiency virus–related thrombocytopenia. Attempts were made to elevate his platelet count (from 32 to 50 × 10^9/L) using zidovudine (AZT, Retrovir), steroids, and intravenous immune globulin, without success.

We performed an uneventful aortic valve replacement in this patient, using high-dose aprotinin according to the protocol of the cardiac surgery department of Hammersmith Hospital, London. After neutralization of the heparin with protamine, the operative field was dry, and the patient was closed. In the immediate postoperative period, 150 mL of blood drained from his pericardial and mediastinal chest tubes. He was reexplored and no surgical source was found for his bleeding. His fibrinogen level was found to be 106 mg/dL. He received 35 U of cryoprecipitate and hemostasis was thereafter satisfactory. The total postoperative chest tube drainage including serosanguinous heart failure fluid was 1,200 mL. The platelet count three hours after bypass was 175 × 10^9/L. Two days later, it had returned to 50 × 10^9/L.

The administration of aprotinin was done using a personal Investigational New Drug exemption obtained from the Food and Drug Administration for use in this specific case, and the aprotinin was supplied by Miles Laboratories. The patient had a benign postoperative course and was discharged on the tenth postoperative day.

We feel that although this is but an anecdotal case, it may show the potential of aprotinin in patients with immune complex thrombocytopenia.

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Management of Sternal Tumors

To the Editor:

We read with interest the article by Gabbay and associates [1] on the management of sternal tumors. We fully agree with the conclusion that all sternal tumors should be biopsied before attempted surgical resection. This approach is accepted nowadays by almost all thoracic surgeons [2, 3]. The references [4, 5] advocating resection without biopsy are not recent; Revitch, in his chapter on the thoracic wall in *Gibbon's Surgery of the Chest* [6], advocates "unequivocal histologic diagnosis by needle biopsy or open biopsy in cases of myeloma or Ewing's sarcoma." Therefore the use of the term *controversies* in the title of the article is somewhat misleading.

Furthermore, Gabbay and associates do not mention whether urine analysis, protein electrophoresis, and bone marrow aspiration, which could identify multiple myeloma, were performed. A solitary plasmacytoma may also cause hypergammaglobulinemia. In the past year we successfully diagnosed 2 cases of isolated plasmacytoma with the aid of a Tru-cut needle biopsy.

As to the reconstruction performed in the patient reported by Gabbay and associates, the use of methylmethacrylate undoubtedly offers a superb cosmetic result; on the other hand, the functional deficit after resection of the manubrium and adjacent cartilages is relatively limited. In our experience of several cases, mesh reconstruction with or without rib or metal strip reinforcement covered by muscle allowed extubation within 24 hours.

In conclusion, we believe that the preoperative evaluation was incomplete, the operation not indicated, and the magnitude of reconstruction too big for this particular case.

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Reply

To the Editor:

My colleagues and I welcome the comments of Drs Yellin and Lieberman. Additionally, we are happy to hear of their agreement with our conclusion. However, we differ regarding the absence of "controversies" to our approach. In fact, during the presentation of our paper at the 1988 meeting of The Society of Thoracic Surgeons, a surgeon from Walter Reed Army Medical Center strongly supported the approach of excisional biopsy of chest wall tumors [1]. Seemingly, Yellin and Lieberman yield to the existence of controversy when they refer to this approach as being accepted by "almost" all thoracic surgeons.

In our case, all laboratory tests performed on admission did not suggest multiple myeloma, including a negative result for Bence Jones protein. Consequently, protein electrophoresis was not performed. As to their comments on chest wall reconstruction, "controversies" certainly do exist. Although they have no doubts that the methylmethacrylate offers superb cosmetic results, it is certainly not an accepted modality of prosthetic reconstruction (although we tend to agree with them). Concerning the functional deficits following resection of the manubrium and both clavicles, as in our patient, we are unaware of any large comparative study that shows that the functional deficits are "relatively limited" or what resultant short-term and long-term complications may exist. The literature shows that some patients have had to be intubated up to 1 week postoperatively. Additionally reported are changes in long-term lung compliance and a tendency for recurrent pneumonia.

Drs Yellin and Lieberman did not discuss other "controversies" presented in our article. However, issues such as election for partial or complete sternum removal in the case of malignancy remain unanswered. Our conclusion clearly stated the rationale for the procedure. Reconstruction was simple and resulted in excellent functional and cosmetic results. We respectfully disagree with the conclusions of Yellin and Lieberman.

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Reference

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Repair of Sternal Dehiscence Using a Harrington Compression System

To the Editor:

We write this letter in response to the article by Miller, Johnson, and Naifeh titled "Repair of sternal dehiscence using a Harrington compression system" [1]. The patient cited in this article is very familiar to us. Five months after this Harrington compression system was installed, he was referred to us with serous fluid draining intermittently from his chest tube site. He was experiencing constant pain, which was exacerbated with each respiratory cycle due to instability of his sternum. The rocking of the inferior Harrington rod with resultant periosteal "grating" by the hooks caused excrutiating pain.

Review of the patient's medical record revealed that after the initial operation for removal of his Greenfield filter, the patient left the hospital with intermittent serous drainage from his chest tube site, as well as a low-grade temperature of "unknown origin." Subsequently, the patient manifested sternal dehiscence that was managed initially with conventional debridement and rewiring, as described. This broke down approximately 7 months after the Greenfield filter removal. The operation described in the article was then performed.

We would like to correct an error in the article and then discuss some aspects of this case.

The patient was admitted to the cardiothoracic service *5½ months* after placement of the Harrington compression system. At that admission, the patient's sternum was grossly unstable. He related a history of sternal instability for several weeks with intermittent serous drainage from chest tube sites. The reported *7-month* follow-up would have been impossible.

Due to his large size, multiple medical problems, and high left above-the-knee amputation which limited him to wheelchair mobility, he presented a perplexing problem. In conjunction with our plastic surgery service, we planned and undertook the following procedures. (1) All hardware was removed, including the Harrington compression system and sternal wires. (2) A radical debridement of the sternum into the costochondral junction was completed. (3) Both pectoralis major muscles were used for reconstruction of the anterior chest and to fill the dead space resulting from the debridement and (4) primary skin closure was accomplished. The patient tolerated the procedure well, and has recovered without any further complications.

The limiting factor in reporting him completely recovered is rehabilitation. His mobility is dependent on upper body strength, and this reconstruction has removed pectoralis major function. He has required extensive physical therapy in an attempt to return him to his normal high level of function and independence. At present, he is undergoing physical therapy in the swimming pool. His shoulder girdle is being retrained to meet his needs for activity. He has returned to work intermittently. His wounds have remained healed, his anterior chest is nearly completely stabilized, and he is pain free. He is very pleased with the results, as are we. We had planned to report this aggressive reconstruction and rehabilitation once it was completed, but unfortunately, the article by Miller and associates has forced us to respond before completion of what we feel is adequate follow-up in this patient.

We regret to speak out so strongly against individuals who have reported what they think is a good technique, but it is our feeling that acute and chronic osteomyelitis must be handled very aggressively, with radical debridement and reconstruction with well-vascularized local muscle flaps. With the use of these muscle flap techniques [2, 3] this difficult reconstruction problem can be managed effectively with excellent results. The references cited by our colleagues in their article supporting Knodt rods, if reviewed carefully, further support *not* using this technique in the face of infection. In the article by Hicks and associates [4], 3 patients treated with rods had infection; one of these failed treatment, and the rods had to be removed. A 33% failure rate in a technique of this nature should not be cited as acceptable.

We treat sternal instability and sternal dehiscence aggressively. If initial radical debridement and sternal rewiring do not succeed, we proceed immediately to local muscle flap reconstruction in conjunction with our plastic surgery service. We have been very pleased with our results as this case illustrates. The difficulties of rehabilitation in patients without this individual's other disabilities are minimal, and can be easily compensated by other muscle groups. This patient raises an interesting dilemma due to his dependence on his upper body strength for mobility secondary to his left above-the-knee amputation. We appreciate the opportunity to respond to this letter and to give follow-up on this very complex patient.

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Reply

To the Editor:

As an orthopaedic surgeon, my involvement with this case was to provide some form of rigid internal fixation stronger than 18-gauge wires for a difficult sternal dehiscence. I then assisted my thoracic surgical colleague in implementing the device in this particular patient. I personally reviewed this patient at 3 to 4 months postoperatively, at which time he was doing well and the roentgenogram that was published in *The Annals* was taken. All subsequent follow-up of this patient was done by others. At the time of his operation, no osteomyelitis of the sternum was found. I can only speculate that because of the number of procedures that this patient had undergone that a bone infection developed after insertion of the Harrington system. It is well established that bone may not heal in the presence of an infection, and I suspect that this is exactly what happened in this case. I feel that the Harrington compression system is a good method of dealing with difficult sternal problems and in the absence of infection would probably result in solid healing. In the presence of infection and a pseudarthrosis, of course the hardware would have to be removed and more extensive procedures as outlined by Dr Cochran and Dr Rohrich would be necessary. I welcome their comments and their follow-up of this interesting problem.

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Direct Access to the Paravertebral Space at Thoracotomy

To the Editor:

We published a technique of continuous extrapleural intercostal nerve block in *The Annals* in October 1988 [1]. We would like to bring attention to an improvement in technique that allows more precise placement of the catheter tip within the paravertebral space.

The parietal pleura is raised from the posterior chest wall as far as the vertebral bodies as described in our original publication. At this point, instead of laying the catheter alongside the vertebral bodies, we make a small defect in the extrapleural fascia using Lahey's forceps and pass the tip of the catheter directly into the paravertebral space through this defect (Fig 1). In this way two or three centimeters of catheter lie within the space itself, which is well perfused through the side holes in the catheter. The pleura is reattached, and the paravertebral space is infused with 0.5% bupivacaine for four postoperative days as previously described.

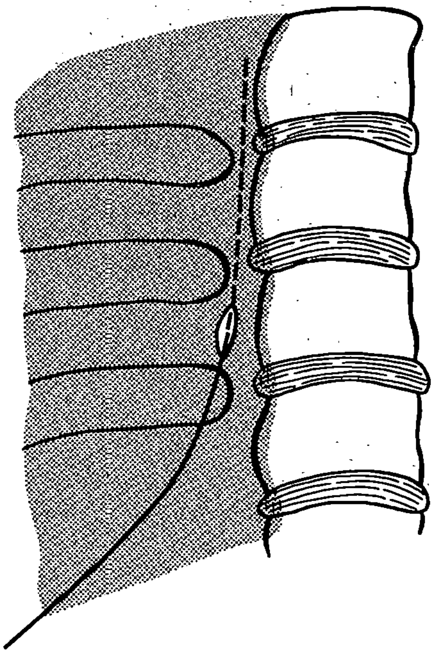

Fig 1. The extrapleural fascia (stippled) *is exposed by raising the parietal pleura from the posterior chest wall. The epidural catheter is lying within the paravertebral space* (broken line), *introduced through the small defect.*

This modification ensures accurate location of the paravertebral space and prevents the catheter from becoming dislodged during subsequent closure of the thoracotomy.

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Reference

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CORRECTION

"Thrombectomy of the Björk-Shiley Prosthetic Valve Revisited: Long-Term Results"

by C. G. Montero, MD, N. Mula, MS, R. Brugos, MD, R. Pradas, MD, and D. Figuera, MD (Ann Thorac Surg 1989;48:824–8)

The Editorial Office has received a communication from the senior author that indicates the submitted manuscript and subsequent publication carry an incorrect author listing. The correct authors are as follows: C. G. Montero, MD, N. Mula, MS, R. Brugos, MD, G. Tellez, MD, and D. Figuera, MD.

Notes From the Thoracic Surgery Directors Association

The Thoracic Surgery Directors Association (TSDA) met in Baltimore, MD, on September 9, 1989.

The principal subject of the meeting was a symposium on thoracic oncology training directed by Dr Martin McKneally. Dr McKneally reviewed a survey of program directors relating to thoracic oncology educational efforts. Considerable variation in thoracic oncology curricula was revealed. Participants in the symposium represented programs that have demonstrated excellence in thoracic oncology training: Dr Nael Martini, Memorial Sloan-Kettering Institute for Cancer Research, Dr John Niederhuber, The Johns Hopkins Hospital, Dr Harvey Pass, National Cancer Institute, and Dr Jack Roth, MD Anderson Cancer Center.

Retiring President Joseph McLaughlin addressed the membership and a summary of his remarks follow:

When I was elected, I looked about for something that we needed to do—something that would impact upon a specialty that some felt had matured and perhaps was narrowing to the point of decline. We know this isn't true, but our behavior all too often supports this perception. We are becoming and training coronary artery surgeons. Not that this is all bad—many of us started out as right upper lobectomy and occasional atrial septal defect surgeons and indeed have had the privilege and the pleasure of growing up with what has been the most dynamic of the surgical subspecialties.

But this growing up, this maturing, this enormous success has, as is so often the case, seen us become complacent, if not in fact, unfortunately all too often in attitude. My son has chosen orthopedics as a career as have so many of our brightest students in recent years. The general surgical residents at the University of Maryland now choose plastic surgery more frequently than any other specialty. One may ask "why?"—and answer "money, good hours, the usual." However, I submit that the most important reason is because of the exciting things that are happening in those specialties: artifical joints, bone lengthening procedures, myocutaneous flaps—all from active and ongoing clinical and basic research.

Thus it seemed to me that my tenure should have as part of its thrust the stimulation of you, the leaders of our profession, to rejuvenate both by attitude and by deed those basic factors so necessary to continued growth, namely, laboratory and clinical investigation.

Accordingly, last year at this session, my confrere from Baltimore, Bruce Reitz, chaired a symposium on research. Paul Ebert supplied us with the National Institutes of Health data on grants and proposals. It turns out that thoracic surgeons do reasonably well in this regard—not as well as in the 1960s when grant money was far less competitive, but much better than I had thought. But also it was apparent that these activities are concentrated in relatively few places, and most frequently are carried out in association with basic scientists and other clinicians in associated specialties. We have a number of PhDs in our department and plan to take on more; a busy thoracic surgeon, even one with great academic bent, doesn't have the time—or for that matter, the training—necessary to engage in basic work. Therefore, this must be addressed by people who are dedicated to the laboratory. Our young people, our residents and junior staff must be provided with the opportunity to work in this environment, to learn the basic techniques and perhaps most importantly to learn what a modern research lab is capable of doing.

Around the turn of the century Claude Bernard is purported to have said that it is a pity that there is nothing left to discover. There is much to discover—much to discover that will have a significant impact upon our specialty. But like everything else, one has to work at discovery. One has to be dedicated to discovery. I urge every one of you to dedicate a part of your time to discovery, to providing the proper environment for research and clinical investigation in your programs. Our future as a specialty depends upon it.

Today we heard about thoracic oncology. My second thrust was in part addressed by the panel this afternoon. We must expand our horizons. To do so we must first examine what constitutes our discipline. When I returned to Baltimore in 1965 I did many vascular procedures—carotid artery endarterectomies, subclavian bypasses, even abdominal aneruysms—now much of which is done by general vascular surgeons, but neurosurgeons also perform carotid and other extracranial vascular procedures and plastic surgeons perform vascular anastomosis for free flaps. Similarly, general surgeons increasingly are performing thoracic procedures. We have made it technically easy with staplers. Pulmonary specialists perform bronchoscopy and interpret pulmonary function studies. Intensivists care for these patients postoperatively.

Some years ago one couldn't be a thoracic surgeon unless one had great expertise in the treatment of lung and esophageal and other thoracic cancers. Thoracic trauma—what is our role in this era of traumatologists? Supportive angioplasty—who runs the pump? All of the foregoing and much of what has developed from it was once the domain of thoracic surgeons. We have neglected a significant part of this domain and, worse, have seen it developed by other specialists. Why has this occurred? Complacency, neglect, lack of time, the tremendous commitment to coronary surgery, money—lots of reasons. I submit that none of these reasons need deter us from a necessary reexamination and rejuvenation of our specialty. This is already starting to happen. Our major societies are addressing the question of vascular surgery. There will be a luncheon meeting of an ad hoc committee directed at training academically oriented general thoracic surgeons this Monday. All to the good, but we as the thoracic surgery program directors should and must be leaders in this reexamination and redirection. Hopefully, these remarks today will stimulate some of you to look at your programs and to reexamine what your thrust for the future will be.

Most of us have made our names, have gotten our jobs because of heart surgery. But our horizons must be broader than this. Most importantly we must expand our horizons with new knowledge that *we* create. Perhaps most importantly of all we must rededicate ourselves to developing the environment to create and utilize this new knowledge.

Dr Benson Wilcox, Chairman of the Nominating Committee, nominated Dr Stan Nolan of Charlottesville for Councillor-at-Large and Dr Martin McKneally of Albany for President-Elect. A unanimous ballot was cast. Dr Nolan's 4-year term commenced immediately. Dr McKneally will serve as Vice-President to President William Gay, Jr, for 2 years before assuming the presidency.

Gordon F. Murray, MD
Secretary-Treasurer

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PRELIMINARY PROGRAM

1990 INTERIM MEETING
THE SOCIETY OF THORACIC SURGEONS

September 21–23, 1990
Hyatt Regency Chicago, Chicago, Illinois

Friday, September 21, 1990

1:00 **Welcoming Remarks**
W. Gerald Rainer, President

Introduction to Program
Richard P. Anderson, Secretary and Interim Meeting Program Chairman

1:10–2:45 **Medico-Legal Session**
Moderator: Thomas D. Bartley, Chairman, Committee on Medico-Legal Affairs

STS Professional Liability Survey
Thomas D. Bartley

The AMA/Specialty Society Medical Liability Project
James S. Todd
Acting Executive Vice President, American Medical Association

California Tort Reform
Winfield J. Wells
Member, Committee on Medico-Legal Affairs

A Trial Lawyer's Perspective
Joseph D. Jamail
From the firm of Jamail and Kolius, Houston, TX

INTERMISSION

3:30–5:00 **Health Care in America**
Moderator: Richard P. Anderson

Health Care Needs—The Consumer Viewpoint
Robert E. McGarrah, Jr
Director for Public Policy, American Federation of State, County, and Municipal Employees

Health Care—A Political Perspective
The Honorable Richard D. Lamm
Professor, Leo Block Chair, Director, Center for Public Policy and Contemporary Issues, The University of Denver; Governor of Colorado, 1975–1987

The Health Care Industry
Austin Ross
Vice President and Executive Administrator, Virginia Mason Medical Center; Clinical Professor, University of Washington School of Public Health and Community Medicine; Chairman, Ad Hoc Committee on Physician Involvement, American Hospital Association; Consultant, W.K. Kellogg Foundation

Saturday, September 22, 1990

7:00–11:00 Two workshops will address reimbursement and coding.

7:00–8:30 **Physician's Breakfast Session**
This session will feature a representative from HCFA discussing recent changes affecting cardiothoracic reimbursement.

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9:00–11:30 **Coding Workshop for Office Personnel** (Physicians welcome)
Experts on CPT-4 and ICD-9 will explain the two coding systems and answer questions related to cardiothoracic billing.

8:30–9:45 **Plenary Session**
Moderator: W. Gerald Rainer

The Canadian Experience
Hugh E. Scully
Member, Committee on Physician Reimbursement

Thoracic Surgery Initiatives
George E. Miller, Jr
Chairman, Committee on Physician Reimbursement

INTERMISSION

10:30–12:00 **Plenary Session**

Paul B. Ginsburg
Executive Director, Physician Payment Review Commission
Washington, DC

Monica Noether
Senior Economist, Abt Associates, Inc
Cambridge, Massachusetts

Glenn R. Markus
STS Consultant on Physician Reimbursement and Government Relations
Health Policy Alternatives, Inc
Washington, DC

12:00–1:30 Luncheon: Invited Speaker
The Honorable Louis W. Sullivan
Secretary, Department of Health and Human Services

AFTERNOON SCIENTIFIC PROGRAM

2:00–5:45 **THREE PARALLEL SCIENTIFIC SESSIONS**

General Thoracic Surgery

2:00 Management of Stage IIIA Lung Cancer
Robert J. Ginsberg, New York, NY

2:20 Management of Barrett's Esophagus
F. Henry Ellis, Jr, Burlington, MA

2:40 Esophageal Diverticula
Mark B. Orringer, Ann Arbor, MI

3:00 Pulmonary Resection for Metastatic Disease
Thomas R. J. Todd, Toronto, Ont, Canada

INTERMISSION

4:10 Multimodality Treatment of Cancer of the Esophagus
Joseph I. Miller, Atlanta, GA

4:30 Mesothelioma
Nael Martini, New York, NY

Parallel Scientific Sessions, continued

4:50 Pancoast Tumor
Harold C. Urschel, Jr, Dallas, TX

5:10 Management of Achalasia
Mark K. Ferguson, Chicago, IL

Adult Cardiac Surgery

2:00 Challenges for Cardiac Transplantation in the Nineties
Bartley P. Griffith, Pittsburgh, PA

2:20 Heart-Lung Transplantation—1990
Bruce A. Reitz, Baltimore, MD

2:40 Current Status of Mechanical Circulatory Support
D. Glenn Pennington, St. Louis, MO

3:00 Cardiac Arrhythmia Surgery: Current Status and Future Directions
James L. Cox, St. Louis, MO

INTERMISSION

4:10 Surgery for Congenital Heart Disease in Adults
James K. Kirklin, Birmingham, AL

Valve Selection for Valve Replacement

4:30 Mechanical Valves
Cary W. Akins, Boston, MA

4:50 Bioprosthetic Valves
William R. E. Jamieson, Vancouver, BC, Canada

5:10 Homograft Valves
Robert B. Karp, Chicago, IL

5:30–5:45 Panel Discussion: Questions and Answers

Pediatric Cardiac Surgery

2:00 Fontan Procedure: Techniques and Results With Tricuspid Atresia and Complex Single Ventricle
Gordon K. Danielson, Rochester, MN

2:20 The Arterial Switch Operation for Simple and Complex Forms of Transposition
Edward L. Bove, Ann Arbor, MI

2:40 Surgical Management and Results in Infants with Pulmonary Atresia With Intact Ventricular Septum
William G. Williams, Toronto, Ont, Canada

3:00 Surgical Management of Coarctation in Infants and Children
Ralph D. Siewers, Pittsburgh, PA

INTERMISSION

4:10 Aortic, Mitral, and Pulmonary Valve Repair in Infants and Children
Aldo R. Castaneda, Boston, MA

4:30 Repair of Truncus Arteriosus in Infants
Kevin Turley, San Francisco, CA

4:50 Surgical Management of Tetralogy of Fallot
Vaughn A. Starnes, Stanford, CA

5:10 Anomalous Origin of the Left Coronary Artery From the Pulmonary Artery
John W. Brown, Indianapolis, IN

Sunday, September 23, 1990

7:00–10:30 **Two sessions on STS National Database**

7:00–8:00 **Breakfast Session for Members and Guests**
Presentation and discussion of collated data on adult cardiac and lung cancer surgery from STS National Database

8:30–10:30 **Workshop**
Detailed presentations, hands on demonstration, and teaching of use of STS National Database designed for surgeons, clinical research coordinators, physician assistants, and office personnel of current and prospective users.

8:00–10:00 **PLENARY SESSION**

8:00 **Lung Transplantation**
Joel D. Cooper, St. Louis, MO

8:40 **Hypoplastic Left Heart Syndrome**
Moderator: Aldo R. Castaneda, Boston, MA

Surgical Repair
William I. Norwood, Jr, Philadelphia, PA

Cardiac Transplantation
Constantine Mavroudis, Chicago, IL

9:20 **Coronary Revascularization**

Angioplasty—Current Indications and Results
Spencer B. King, Atlanta, GA

Coronary Bypass—Current Indications and Results
Floyd D. Loop, Cleveland, OH

10:45–12:00 **THREE PARALLEL HOW-TO-DO-IT SESSIONS**

General Thoracic Surgery

10:45 Management of Infected Median Sternotomy Wounds
Geoffrey M. Graeber, Morgantown, WV

11:00 Intrathoracic Lung Cancer Staging
G. Alexander Patterson, Toronto, Ont, Canada

11:15 Sleeve Lobectomy
L. Penfield Faber, Chicago, IL

11:30 Ivor-Lewis Esophagogastrectomy
Victor F. Trastek, Rochester, MN

11:45 Postpneumonectomy Empyema
Peter C. Pairolero, Rochester, MN

Adult Cardiac Surgery

10:45 Revascularization With the Gastroepiploic Artery
Bruce W. Lytle, Cleveland, OH

11:00 Retrograde Cardioplegia
W. Randolph Chitwood, Jr, Greenville, NC

11:15 Adult Homograft Aortic Valve Replacement: Surgical Challenges Requiring a Spectrum of Techniques for Solution
Richard A. Hopkins, Washington, DC

11:30 Mitral Valve Repair—1990
Delos M. Cosgrove III, Cleveland, OH

11:45 Treatment of Aortic Dissection—Updated
E. Stanley Crawford, Houston, TX

Parallel How-to-Do-It Sessions, continued

Pediatric Cardiac Surgery

10:45 Postoperative Management of Pulmonary Hypertension in Infants and Children Including ECMO
James A. Alexander, Gainesville, FL

11:00 Atrioventricular Septal Defect Repair in Infancy
Frank M. Midgley, Washington, DC

11:15 What to Do If It Can't Be Fixed: Closed Palliative Procedures
Anthony L. Moulton, Omaha, NE

11:30 Surgical Ablation of Arrhythmias in Children
David A. Ott, Houston, TX

11:45 Cryopreserved Allograft Valve Conduits and Vascular Tissue in the Repair of Complex Congenital Heart Disease
David R. Clarke, Denver, CO

12:00 ADJOURNMENT

1991 Annual Meeting of The Society of Thoracic Surgeons

The 1990 meeting is an Interim Meeting to be held at the Hyatt Regency Chicago in Chicago, Illinois, September 21–23, 1990. No abstracts are solicited for that program.

The Twenty-sixth Annual Meeting will be held at the San Francisco Moscone Center in San Francisco, California, February 18–20, 1991.

APPLICATION FOR MEMBERSHIP

(Deadline: September 1, 1990)

Requests for application forms for membership in The Society of Thoracic Surgeons should be addressed to James L. Cox, MD, Chairman, Membership Committee, The Society of Thoracic Surgeons, 111 E Wacker Dr, Chicago, IL 60601.

The completed application forms and supporting letters must be in the hands of the Membership Committee by **September 1, 1990,** for the applicant to be considered for election at the meeting in February 1991.

CALL FOR ABSTRACTS

(Deadline: September 12, 1990)

Abstracts for papers to be presented at the 1991 Annual Meeting are now being accepted. The deadline for receipt of abstracts is September 12, 1990. An original and one copy should be submitted, using the official Society abstract form (on verso of this page), to Alexander S. Geha, MD, and Robert A. Guyton, MD, Co-Chairmen, Program Committee (Abstracts), The Society of Thoracic Surgeons, 111 E Wacker Dr, Chicago, IL 60601. Additional forms are available upon request from The Society. Abstracts must summarize an original contribution not presented or accepted for presentation elsewhere. Abstracts must be in the hands of the Program Committee by **September 12, 1990.** Abstracts received after the deadline will not be considered. It is requested that the covering letter indicate the author who is to receive notice of acceptance or rejection and the author who will present the paper.

The Thoracic Surgery Directors Association (TSDA) has established a Resident Presentation Award for 1991. Potential TSDA Resident Award abstracts must represent original work by the resident, who is to be first author and presenter. TSDA Resident Award abstracts may be directed to the regular Scientific Session or the Research Forum.

Essayists are reminded that according to the bylaws of The Society, all papers accepted for presentation: (1) must be submitted in *triplicate* to the Secretary at the time of presentation or to the Editor of *The Annals of Thoracic Surgery* prior to presentation; and (2) become the property of The Society if they are accepted for presentation.

It is well recognized that abstracts frequently belie their content by virtue of poor organization, and as a result valuable contributions to the Scientific Sessions may be unrecognized. It is important that methods and results be clearly defined in the abstract; therefore, general statements such as "results will be presented" are not acceptable. When a procedure or method is stated to have "advantages," those advantages should be defined. Abstracts that simply summarize results without defining objectives and methods are equally undesirable.

Speakers for accepted papers are limited to ten minutes.

Instructions for Abstracts

The official abstract form printed on verso of this page and available from The Society, or photocopies thereof, must be used. **Authors must categorize the principal subject of their abstract by checking the appropriate box.**

If the author wishes to submit the abstract for consideration for the Thoracic Surgery Directors Association Award competition, the *additional* appropriate box must be checked. Also, the name of the Program Director must appear on a TSDA Resident Award abstract.

1. Full names of authors must appear on the abstract.
2. Institution or address is to be given, but omit titles, degrees, and appointments.
3. Abstracts must be limited to 250 words and must be typed double-spaced.
4. When percentages are used, the absolute numbers of derivation must be stated.
5. The body of the abstract should include:
   a. A brief statement of the purpose of the study being presented, with reference to the current state of research in the field.
   b. The methods of study or experimental approach clearly, but briefly, defined.
   c. A summary of the results and/or conclusions of the study, including sufficient details to support those conclusions.
   d. A statement concerning the significance of the work and its possible implications for further research.
6. The form must be signed by the senior author attesting that the material will not be presented elsewhere if accepted for the scientific program nor published elsewhere if accepted for publication in *The Annals of Thoracic Surgery.*

In discussion of papers, The Society encourages a healthy spirit of constructive critical review and rebuttal by comments pertinent to the content of the paper being presented. Presentation of discussion in the form of "secondary papers" will not be welcomed.

CALL FOR SURGICAL FILMS OR VIDEO TAPES

(Deadline: September 1, 1990)

The popular surgical movie program will again be presented at the San Francisco meeting. The Program Committee hereby solicits the submission of surgical films, which may portray a surgical procedure or simply a technical maneuver that the surgeon thinks will be of interest to The Society. The film may not exceed ten minutes in length. Sound movies are acceptable but not necessary; however, live narration by the principal surgeon is a requirement for participation in the movie night program. The movies should be on 16-mm film or ¾-inch U-Matic video tape. The deadline for submission is **September 1, 1990.** Films and video tapes also should be sent to Alexander S. Geha, MD, and Robert A. Guyton, MD, Co-Chairmen, Program Committee, at The Society of Thoracic Surgeons, 111 E Wacker Dr, Chicago, IL 60601.

Richard P. Anderson, MD
Secretary

For Committee use only:

THE SOCIETY OF THORACIC SURGEONS

Abstract Form

For Committee use only:
Rating: (Highest 1)

1☐ 2☐ 3☐ 4☐ 5☐

LIMIT TO 250 WORDS AND DOUBLE-SPACE

TITLE

AUTHOR(S)

ADDRESS

PLEASE CHECK ONE APPROPRIATE CATEGORY

- ☐ **Pulmonary**
- ☐ **Esophagus**
- ☐ **Chest wall Mediastinum**
- ☐ **Trauma**
- ☐ **Transplantation**
- ☐ **Heart**
- ☐ **Great vessels**
- ☐ **Valves**
- ☐ **Congenital**
- ☐ **Coronary**
- ☐ **Electrophysiology**
- ☐ **Myocardial protection**
- ☐ **Extracorporeal circulation**
- ☐ **Endoscopy**
- ☐ **Vascular surgery**
- ☐ **Research, Cardiac**
- ☐ **Research, Congenital Cardiac**
- ☐ **Research, Transplantation**
- ☐ **Research, General Thoracic**

☐ **PLEASE CHECK IF ALSO SUBMITTED FOR THE THORACIC SURGERY DIRECTORS ASSOCIATION AWARD**

Program Director's Name:

I acknowledge that the material will not be presented elsewhere if accepted for presentation on The Society of Thoracic Surgeon Scientific Program. I further understand that the Editorial Board of *The Annals of Thoracic Surgery* has the right of first refus for publication of this material.

Signature of Senior Author

Information About Membership in The Society of Thoracic Surgeons

The Society of Thoracic Surgeons (STS) is the largest organization of its kind devoted primarily to the practice of thoracic and cardiovascular surgery. The major purpose of The Society is educational, with the aim of providing thoracic and cardiovascular surgeons from both academic and private practice environments a forum for presentation and discussion of scientific topics. As outlined in the original bylaws of The Society, this educational emphasis is designed to accomplish the broader goal of improving the quality and practice of thoracic and cardiovascular surgery as a specialty.

The STS recently celebrated its 25th Anniversary, having been founded in 1964. Since its inception, The Society has grown continually so that at present the membership includes the majority of board certified thoracic surgeons in the United States as well as thoracic and cardiovascular surgeons from 45 other countries.

In addition to an annual scientific meeting, the STS has more than 20 committees working on diverse matters related to improving thoracic and cardiovascular surgery as a specialty. Examples of these committees are: Standards and Ethics, Government Relations, Medico-Legal Affairs, Surgical Standby for PTCA, Physician Reimbursement, CPT-4 Advisory, Education and Resources, and Cardiothoracic Surgical Practice Guidelines. The Committee to Analyze Circulatory Assist Devices and Artificial Hearts sponsors topical meetings (next meeting, November 15–17, 1991, in San Francisco). The Committee to Develop a National Database for Thoracic Surgery plans to implement the first comprehensive database program for thoracic and cardiovascular surgeons in the United States; it will be offered to members in the near future.

There are five types of membership in The Society of Thoracic Surgeons: active, senior, honorary, international, and candidate.

Active Member Applicants for active membership must be surgeons whose practice is devoted primarily to the field of thoracic and cardiovascular surgery.

QUALIFICATIONS: Applicants must be Diplomates of the American Board of Thoracic Surgery or Fellows in The Royal College of Surgeons of Canada, and be established in the practice of thoracic and/or cardiovascular surgery for a minimum of three years. There must be sponsoring letters from three active STS members.

Senior Member Any active member or international member, upon reaching the age of sixty-five years, shall automatically become a Senior Member.

Honorary Member Honorary Membership shall be reserved for distinguished persons worldwide who have achieved prominence in the field of thoracic and cardiovascular surgery and have contributed to its advancement. Honorary members shall be elected by a majority vote of The Society at its annual business session upon recommendation of the Council of the STS.

International Member Members from countries other than the United States and Canada were previously accepted only as active members. Because of differences in training and practice, the application process for foreign members was often complex and time consuming. To allow for greater participation by thoracic and cardiovascular surgeons from other countries, the bylaws have been revised to provide for a category of International Member, which is the category for all members outside the United States and Canada.

QUALIFICATIONS: Applicants shall reside outside the United States and Canada. Their training in this specialty shall be considered equivalent to that of Active Members. They shall have achieved appropriate certification or recognition of achievements in this specialty in their country and must devote the majority of their professional efforts to the interests of the specialty. Appropriate documentation of professional achievements shall be provided to the Membership Committee.

Candidate Member Candidate membership was created to allow participation of younger surgeons in the time prior to being qualified for active membership.

QUALIFICATIONS: Applicants shall be enrolled in a thoracic and cardiovascular training program accredited by the Residency Review Committee for Thoracic Surgery under the authority of the Accreditation Council for Graduate Medical Education or in a similar program approved for training by The Royal College of Surgeons of Canada. Also, individuals who have completed their training in one of the above programs and are in process of acquiring certification by the American Board of Thoracic Surgery or The Royal College of Surgeons of Canada are eligible for this type of membership.

If you would like to receive an application for membership in The Society, send the request printed below. This year's deadline for receipt of applications is September 1, 1990. We suggest you apply soon as it takes time to process the application. Approved applications will be presented to the membership at the February 1991 Annual Meeting in San Francisco.

REQUEST FOR APPLICATIONS

Please send an application for: ☐ Active membership ☐ International membership ☐ Candidate membership

TO: NAME: ______________________________

ADDRESS: ______________________________

____

TELEPHONE NUMBER: ______________________________

Send to: James L. Cox, MD, Chairman, Membership Committee
The Society of Thoracic Surgeons, 111 East Wacker Drive, Suite 600, Chicago, IL 60601
Telephone: (312) 644-6610; FAX: (312) 938-1214

THE ANNALS OF THORACIC SURGERY
Official Journal of The Society of Thoracic Surgeons and the Southern Thoracic Surgical Association

EVENTS OF INTEREST

Brief notices of meetings, courses, and symposia relevant to cardiothoracic surgery are published as a service. They should be formatted as follows: meeting title, date, place, sponsor, accreditation, director, fee, minimum/maximum attendance, and address/phone for further information. Announcements should be sent at least 4 months in advance of publication to *The Annals of Thoracic Surgery*, 3108 Queeny Tower, Barnes Hospital Plaza, St. Louis, MO 63110-1041; telephone, (314) 361-6084.

Eleventh Annual Scientific Session of the North American Society of Pacing and Electrophysiology, San Diego, California— May 31–June 2, 1990

For information on this meeting, contact the North American Society of Pacing and Electrophysiology, 13 Eaton Court, Wellesley Hills, MA 02181; or telephone (617) 237-1866 (facsimile: (617) 431-1991).

Eighth Annual Uniformed Services University of Health Sciences Cardiothoracic Surgery Symposium, Washington, DC— May 31–June 2, 1990

For information on this meeting, contact Fred H. Edwards, MD, Department of Thoracic Surgery, Walter Reed Army Medical Center, Washington, DC 20307-5001; or telephone (202) 576-1433.

The Society for Vascular Surgery/International Society for Cardiovascular Surgery, Los Angeles, California—June 4–6, 1990

For information on this meeting, contact the Association offices, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330.

Seventh International Congress Cardiostim 90, Nice, France—June 20–23, 1990

Cardiostim gathers several scientific events: Cardiostimulation 90 (cardiac pacing, electrophysiology, defibrillation), International Conference on High-Resolution ECG and Late Potentials, Fifth World Congress on Catheter Ablation, Cardio-Pharmaco 90, and Cardio-Rythmo 90. For information on this meeting, contact Secretariat Cardiostim 90, Department of Cardiac Pacing, Centre Chirurgical Val D'Or, 16, rue Pasteur, 92211 St. Cloud Cedex, France; or telephone (1) 46.02.70.72 (facsimile: (1) 46.02.05.09).

Sixteenth Annual Meeting of The Western Thoracic Surgical Association, Coronado, California—June 20–24, 1990

For information on this meeting, contact The Western Thoracic Surgical Association, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330.

Internal Thoracic Artery for Myocardial Revascularization, Toulouse, France— June 27–29, 1990

For information on this meeting, contact the Secretary of the Symposium, Department of Cardiovascular Surgery, Rangueil University Hospital, 31054 Toulouse Cedex, France; or telephone 61.32.26.51 or 61.32.26.53 (telex: 530 826 F).

Cardiac Arrhythmia Surgery International Seminar I, Geneva, Switzerland— June 27–29, 1990

For information on this meeting, contact Beverly Farrier, ProMedica International, 620 Newport Center Drive, Suite 575, Newport Beach, CA 92660; or telephone (714) 640-5870 (facsimile: (714) 759-6911).

New Devices for the Cardiac and Peripheral Vascular Laboratories, Orlando, Florida— July 2–6, 1990

This meeting is sponsored by the Pittsburgh Vascular Institute and the Pittsburgh Heart Institute of Shadyside Hospital, Pittsburgh, PA, and is accredited for 15 hours in category 1 of the Physician's Recognition Award of the AMA. For information on this meeting, contact Shadyside Hospital, New Devices Technology Workshop, 5230 Centre Ave, Pittsburgh, PA 15232; or telephone (412) 622-1051.

Seventh Congress of the European Society of Biomechanics, Aarhus, Denmark—July 8–11, 1990

For information on this meeting, contact Seventh Congress of ESB, Biomechanics Laboratory, Orthopaedic Hospital, Randersvej 1, 8200 Aarhus N, Denmark; or telephone 45-616-7500, extension 4649.

Myocardial Preservation Into the 21st Century, Oxford, England—August 12–15, 1990

For information on this meeting, contact Beverly Farrier, ProMedica International, 620 Newport Center Drive, Suite 575, Newport Beach, CA 92660; or telephone (714) 640-5870 (facsimile: (714) 759-6911).

Thirty-ninth Annual Meeting of the Scandanavian Association for Thoracic and Cardiovascular Surgery and the Scandanavian Society for Extracorporeal Technology, Reykjavík, Iceland—August 29–31, 1990

For information on this meeting, contact G. Ólafsson, MD, Chairman, Department of Thoracic and Cardiovascular Surgery, Landspítalinn, University Hospital, Reykjavík, Iceland.

Thirty-ninth Congress of the European Society for Cardiovascular Surgery, Budapest, Hungary—September 9–12, 1990

For information on this meeting, contact Budapest Convention Centre, H-1444, PO Box 233, Budapest XI, Jagello u 1-3, Hungary; or telephone (361) 852-507 (facsimile: (361) 665-636; telex: 22-7717).

Fourth Annual Meeting of the European Association for Cardio-thoracic Surgery, Naples, Italy—September 17–19, 1990

For information on this meeting, contact the Organizing Secretariat, MCM, Rione Sirignano, 5, 80121 Naples, Italy; or telephone (0)81/7611085-7611277 (facsimile: (0)81/664372; telex: 710626).

Second Annual Meeting of the International Society for Applied Cardiovascular Biology, Venice Lido, Italy—September 17–20, 1990

For information on this meeting, contact the Congress Secretariat, Kele Teo, San Marco 4930, Venice, Italy.

Interim Meeting of The Society of Thoracic Surgeons, Chicago, Illinois—September 22–23, 1990

For information on this meeting, contact Richard P. Anderson, MD, The Society of Thoracic Surgeons, 111 East Wacker Dr, Chicago, IL 60601-4301; or telephone (312) 644-6610.

Mechanical Circulatory Support 1990, London, England—September 26–28, 1990

For information on this meeting, contact Anne Sumner, Mechanical Circulatory Support 1990, Medical Research Services, 60 Oxford St, Southampton SO1 1DL, Hants, England; or telephone 0703 231068.

Controversies in Cardiovascular Disease: Surgical Versus Medical Treatment, Aalst, Belgium—September 28–29, 1990

For information on this meeting, contact Dr F. Wellens, Department of Cardiovascular Surgery, O.L. Vrouwziekenhuis, Moorselbaan 164, B-9300 Aalst, Belgium; or telephone 053/78 53 53 (facsimile: 053/78 38 76).

CLASSIFIED ADS

Classified ads for positions wanted or available must relate only to the thoracic or cardiovascular surgical specialties. Display space (minimum, ¼ page) is also available through Media for Medicine, Inc, 440 Park Avenue South, New York, NY 10016 (telephone: (212) 684-5540; facsimile: (212) 545-0651). All ads are subject to the approval of the editor, and material deemed inappropriate for publication in *The Annals* will be rejected. If anonymity is desired, please indicate this at the time of submission and a code number will be assigned. All responses received will remain confidential in the publisher's office and will be conveyed to the advertiser shortly after receipt. The charge for all classified advertising is **$1.10 per word per insertion, minimum 20 words.** Abbreviations, dates, initials, post office box numbers, telephone numbers, years, and zip codes are considered one word each. **There is an additional fee of $15.00 per insertion for box number ads.** The copy deadline is 7 weeks prior to publication, eg, for the March issue, copy should be received by the 1st of January. Ad orders should be for a maximum of 6 months; ads may be renewed for up to 6 months at a time. Ad copy should be typed double-spaced and mailed in duplicate to: *The Annals of Thoracic Surgery,* Desk Editorial, Classified Ads, Elsevier Science Publishing Co, Inc, 655 Avenue of the Americas, New York, NY 10010 (facsimile: (212) 633-3695). Make *nonrefundable* check payable to Elsevier Science Publishing Co, Inc.

Please note that *The Annals* does not function as a placement service and cannot provide information on advertised positions. When answering advertisements, please write individually to each advertiser you wish to contact, using the address given in the ad. To respond to an anonymous ad, write to Box Number, *The Annals of Thoracic Surgery,* Elsevier Science Publishing, 655 Avenue of the Americas, New York, NY 10010.

SITUATIONS AVAILABLE

Cardiothoracic surgeon wanted for a busy, expanding, 3-man, private practice group in the Midwest. Applicant must be ABTS eligible or certified. Prefer recent graduate with some background in electrophysiologic surgery. Please send curriculum vitae.

Please respond to Box 189. 189H/G

Cardiothoracic surgeon, BE/BC, wanted to join surgeon in active cardiac/thoracic/vascular surgical practice in northern Alabama community of 80,000 (300,000 population draw). Clinical practice in two community hospitals. Lake/mountain recreation facilities. Generous compensation, future partnership.

Please write Sara Fleming, Business Manager, 801 Gaines Ave, Suite 305, Gadsden, AL 35903; (205) 492-9360. 237L/E

Associate Director for Heart Transplantation is urgently needed for rapidly expanding heart transplantation program in California.

Please respond with curriculum vitae to the Pacific Cardiothoracic Surgery Group, 201 S Alvarado St, Suite 626, Los Angeles, CA 90057. 240L/K

Cardiovascular and thoracic surgeon, board eligible/certified, wanted to join well-established private practice located in Ft. Lauderdale, FL. Send CV.

Please respond to Box 251. 251L/K

Cardiovascular and thoracic surgeon, board eligible or certified, wanted to join another surgeon in private practice in the Southeast. Please respond with curriculum vitae.

Please respond to Box 252. 252L/F

Thoracic-vascular surgeon wanted to join busy practice in Northeast. Board certified or eligible. Interested in practice of vascular and thoracic surgery. Excellent benefits package. Send curriculum vitae.

Please respond to Box 254. 254L/F

Cardiothoracic and vascular surgeon wanted to be first assistant. Twenty years experience preferred. Interested in practice of cardiac, vascular, and thoracic surgery. Excellent benefits package. Send curriculum vitae.

Please respond to Box 255. 255L/F

Cardiothoracic surgeon—to join a 2-man private practice in cardiac, thoracic, and vascular surgery in the Great Lakes region. Must be board certified or eligible. Recent graduates considered. Send curriculum vitae.

Please respond to Box 260. 260L/E

Cardiovascular/thoracic surgeon to complete 3-man group in adult cardiothoracic private practice in Michigan. Must be board eligible or certified. References required.

Please respond to Box 269. 269A/F

Cardiothoracic surgeon, board certified with 2 to 4 years experience in cardiac surgery, wanted to join a growing group of cardiac surgeons, cardiologists, and pulmonologists in the Pacific Northwest. General thoracic and vascular experience necessary. Research opportunities available, academic links possible. Salary leading to full partnership in 2 or 3 years; potential exists for leadership in major cardiac program.

Please send curriculum vitae to John R. Bauman, Administrator at The Thoracic Clinic, PC, 507 NE 47th Ave, Portland OR 97213-2282. 274A/F

Cardiovascular and thoracic surgeon: Position available for a board certified or board eligible to join practice in Northeast involved in adult cardiac, thoracic, and vascular surgery. Association will lead to partnership. Excellent fringe benefits. Please send curriculum vitae.

Please respond to Box 275. 275A/F

Cardiovascular and thoracic surgeon, BC/BE, recently trained to join an established 3-man private practice group in Southeastern coastal capital city and two-university town. Great hunting, fishing, and sports. Submit CV and references.

Please respond to Box 293. 293B/E

Cardiovascular and thoracic surgeon: Position available for board certified or board eligible person to join busy practice involved in adult cardiac, thoracic, and vascular surgery in major New England city. Association will lead to partnership. Excellent fringe benefits. Please send curriculum vitae.

Please respond to Box 291. 291B/G

Cardiothoracic/vascular surgeon, board eligible/certified, wanted to join established group practice in Southeast. Please send curriculum vitae.

Please respond to Box 295. 295C/E

Cardiovascular and thoracic surgeon, BE/BC, to join another physician in a rapidly expanding private practice located in the Southeast. Send curriculum vitae and references.

Please respond to Box 296. 296C/E

Thoracic/vascular surgeon to join small group private practice in medium-sized New England community. BC/BE in thoracic surgery. Prefer C/E for Cert. Special Competence in Vascular Surgery. Recent medical graduate preferred. University-affiliated hospital. Full partnership in 3 years.

Please respond to Box 301. 301C/F

Cardiothoracic/vascular surgeon wanted to join established practice in Ohio. Recent graduate preferred. Please submit curriculum vitae and three references.

Please respond to Box 302. 302C/I

Thoracic, vascular, and general surgeon to join a busy, all boarded, private practice on eastern Long Island. Should be board certified or eligible. Excellent salary and benefits, leading to a full partnership. Send curriculum vitae.

Please respond to Box 303. 303C/E

Thoracic and vascular surgeon, board certified, to join active (noncardiac) thoracic and vascular practice in northern New Jersey. Competitive salary and benefits. Please respond with curriculum vitae.

Please respond to Box 304. 304C/E

Group of two cardiothoracic surgeons seeking third associate. Recent graduate preferred. American Board of Thoracic Surgery certification or eligibility is required. Practice consists of adult cardiac surgery at University-affiliated hospital in the New York metropolitan area.

Please respond to Box 310. 310D/F

Cardiovascular surgeon to become second surgeon in new (two year old) adult cardiac program on west coast of Florida.

Submit CV to Manatee Cardiac Surgeons, PA, 201 4th Ave East #2, Bradenton, FL 34208. 312D/F

Cardiac surgeon wanted to join established private practice in Eastern PA. Primarily adult cardiac. BC/BE required. Please send CV.

Please respond to Box 315. 315D/I

Cardiovascular and thoracic surgeon, board eligible or certified, wanted to join another physician in private practice in Northeast. Immediate availability. Please respond with curriculum vitae.

Please respond to Box 316. 316D/I

Cardiovascular-thoracic surgeon, board certified or eligible wanted for established fast-growing practice in south Texas with modern hospital facilities; primarily adult cardiac surgery, thoracic and vascular. Initial contact should include curriculum vitae, clinical experience, surgical results, and names of references.

Please respond to Box 319. 319D/E

Cardiac surgeon with additional interest and experience in vascular surgery wanted to join large, active group practice in suburban Washington, DC. Prefer recent ABTS-certified surgeon with 2 to 5 years experience.

Please respond to Box 322. 322D/F

Academic Program Director, General Surgery Residency Training Program. MacNeal Hospital, a west suburban Chicago teaching hospital, is seeking an Academic Program Director to lead its General Surgery Residency Training Program. The individual must be board certified in general surgery and a surgical subspecialty. The candidate selected will assume appropriate administrative and teaching responsibilities for the professional and scholarly growth of the department; will also actively participate in patient care and conduct daily rounds with house staff. The ideal candidate has national stature in the field of surgery, with a demonstrated record of excellence in scholarship, clinical service, and academic administration. MacNeal Hospital is a 427-bed facility, accredited to educate 5 General Surgery Residents in an integrated program with the University of Illinois. Academic appointment is required. A competitive salary and benefit package is available.

Candidates should forward a letter of application and CV to Jerry Lee, Vice President, Physician Affairs, MacNeal Hospital, 3249 S Oak Park Ave, Berwyn, IL 60402. 323D/E

Research Associate in Cardiovascular Surgery to work in established, productive, experimental surgery laboratory. Projects in temporary and permanent left ventricular assist systems and related artificial organs. Opportunity to participate in clinical research. Clinical open heart surgery experience essential; research background desirable. Send curriculum vitae.

Please respond to Box 324. 324D/E

Cardiovascular thoracic surgeon—board certified/board eligible and recently trained to join an established, busy, dynamic, and expanding private practice in northwest Indiana. The sphere of practice is approximately 30 minutes from downtown Chicago. Guaranteed excellent starting salary with progressive future growth and comprehensive corporate fringe benefit program.

Write including CV and details of experience and capabilities to PO Box 3066, Munster, IN 46321. 329E/F

Cardiovascular surgeon, board eligible/certified, wanted to join a busy adult cardiac surgery practice in Seattle area. Respond with curriculum vitae and references.

Please respond to Box 330. 330E

Cardiovascular/thoracic surgeon, BC/BE, prefer recent graduate to join 2-man group for private practice, predominently in cardiac surgery in Orange County, southern California. Excellent salary. Send CV, with experience and recommendations.

Please respond to Box 333. 333E

Cardiovascular-thoracic surgeon, BC/BE, needed to join busy expanding private practice in the Midwest. Excellent program, large number of experienced cardiologists. Initial contact should include curriculum vitae and references. Ideal candidate will have results to submit.

Please respond to Box 334. 334E

Excellent opportunity for recent graduate, board eligible or certified, for private practice of cardiac, thoracic, and major vascular cases in Great Lakes area. Include references and curriculum vitae.

Please respond to Box 335. 335E/G

The VA Medical Center West Los Angeles is seeking a full-time cardiothoracic surgeon to join its staff. The VAMC West Los Angeles is affiliated with the University of California, Los Angeles. Qualifications include expertise in cardiac surgery and thoracic procedures as well as a commitment to research and teaching in academic environment. Applicant recruiting deadline is June 1, 1990, with an anticipated starting date of July 1, 1990.

Forward CV and the names and addresses of three references to Brenda L. Cabunoc, Personnel Staffing Specialist, Personnel Service (05C-BC), VAMC West Los Angeles, Los Angeles, CA 90073. The Department of Veterans Affairs is an equal opportunity employer. 336E

Cardiothoracic surgeon needed for expanding program in mid-Michigan. Primarily adult cardiac and thoracic. BC/BE required. Please send CV with references.

Please respond to Box 337. 337E/J

Excellent potential achievable by associating with successful BC cardiothoracic surgeon. Aggressive, regional hospital boasts new open-heart facility. This sprawling river community is located in the Southeast and is ranked as one of the 50 best towns in America. Water sports, cultural arts, and fantastic housing options await you.

For more information, send CV to John Bowers, of Harris Kovacs Alderman, 4170 Ashford-Dunwoody Rd, Suite 490, Atlanta, GA 30319; or call (404) 252-7987, ext. 698, COLLECT. 338E

Cardiovascular and thoracic surgeon: Position available for a board certified or eligible cardiovascular and thoracic surgeon to join a 2-man private practice involved with adult cardiac, tho-

racic, and peripheral vascular surgery in a desirable eastern metropolitan area. Association features competitive salary, excellent fringe benefits, and a goal of early partnership. Please send curriculum vitae.

Please respond to Box 339. 339E/G

Thoracic surgeon: Applications are invited for an active staff position at the University of Alberta Hospitals in the Division of Cardiovascular and Thoracic Surgery of the Department of Surgery. Candidates must have the Fellowship of the Royal College of Physicians and Surgeons of Canada in cardiovascular and thoracic surgery or certification of special competence in thoracic surgery. The position will carry an appropriate clinical rank in the Faculty of Medicine for the University of Alberta and candidates must be willing to participate in the teaching programs of the Faculty and support academic activity. They must also be eligible for registration with the College of Physicians and Surgeons of Alberta. In accordance with Canadian immigration requirements, priority will be given to Canadian citizens and permanent residents of Canada.

Please submit a current curriculum vitae and the names of 3 references by June 30, 1990, to Dr Bryce Weir, Chairman, Department of Surgery, Faculty of Medicine, University of Alberta, 2D2 Walter C. Mackenzie Health Sciences Centre, 8440-112 St, Edmonton, Alberta, T6G 2B7, Canada. The University of Alberta is committed to the principle of equity in employment. 340E

Cardiothoracic surgeon: Excellent opportunity in mid-West for experienced cardiothoracic surgeon to join highly regarded board-certified physician who is Chief of Cardiothoracic Service at 450-bed community teaching hospital. Very active practice in adult open hearts and general thoracic. Private practice association leading to partnership.

Please contact Jeanne Dowrick, Fulton, Longshore and Associates, 527 Plymouth Rd, Plymouth Meeting, PA 19462; telephone (800) 346-8397 or (215) 834-6780. 341E/G

SITUATIONS WANTED

Pediatric and adult cardiovascular surgeon, 37, completed fellowship in major university medical center. Extensive clinical and research experience. Interested in academic position or private practice. Curriculum vitae and references upon request.

Please respond to Box 311. 311D/E

Cardiothoracic surgeon, 34, American, NE university training, 10 months private practice experience, wishes to relocate. ABS, ABTS eligible. Experienced in adult coronary reoperations, ventricular assists, and electrophysiologic surgery. Seeks practice in Southwest/Southeast. Curriculum vitae and references upon request.

Please respond to Box 318. 318D/G

53-year-old cardiac surgeon. Current in vascular and thoracic. Strong academic and private practice background. Established private open heart program 11 years ago. At present position. Numerous publications, board certified, American graduate, and advanced medical degree. Wish to relocate.

Please respond to Box 331. 331E

FELLOWSHIPS

Clinical fellowship in adult cardiothoracic surgery available July 1, 1990. Fellowship experience will include acquired cardiac, general thoracic, esophageal, and pulmonary surgery and diagnostic and laser therapeutic endoscopic procedures. Competitive salary and benefits package offered. Minimum requirement: American Board of Surgery certification or eligibility.

Please send resume to Division of Cardiothoracic Surgery, University of Missouri-Columbia, School of Medicine, One Hospital Drive, Columbia, MO 65212. 332E/F

CARDIOVASCULAR SURGEON NEEDED FOR SAGINAW, MICHIGAN

Two man cardiovascular surgical group, with an active, expanding practice in Eastern Mid-Michigan, has an immediate opening for a cardiovascular surgeon, Board Certified/Board Eligible.

Saginaw, Michigan is the gateway to beautiful northern Michigan and serves as the focal point for a fourteen county health planning area, which encompasses close to 1,000,000 people.

For further information about this exceptional private practice opportunity, please send a CV to:

Ms. Donna Bowden
East Michigan Cardiovascular Surgery, P.C.
100 Harrow Lane
Saginaw, MI 48603

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For details, contact: F. L. Korompai, M.D., Scott & White Clinic, 2401 South 31st Street, Temple, Texas, 76508, 817-774-4910.

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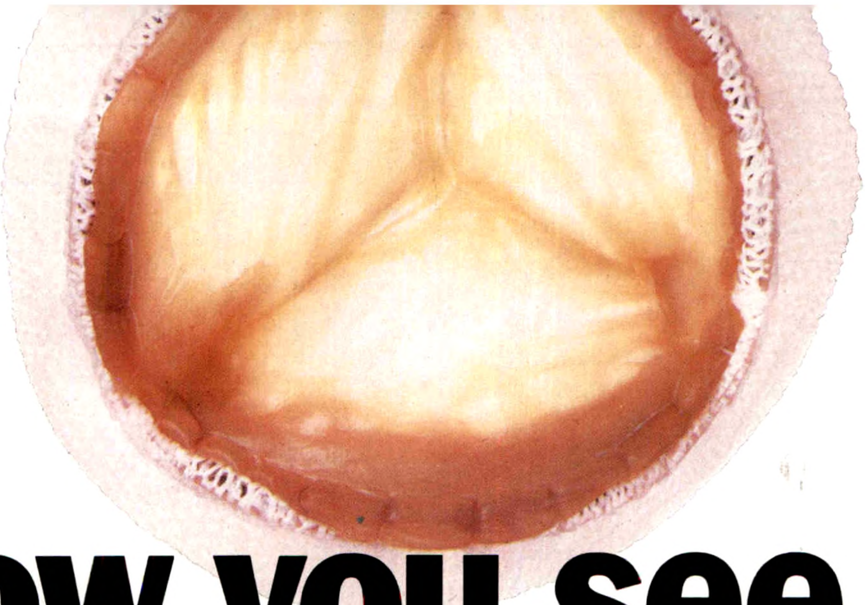

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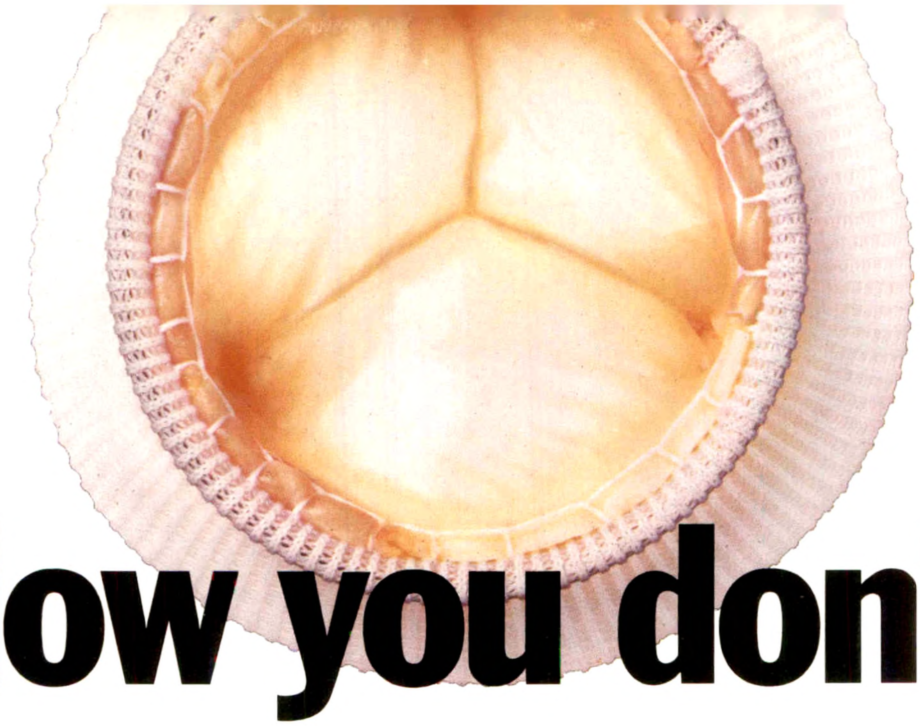

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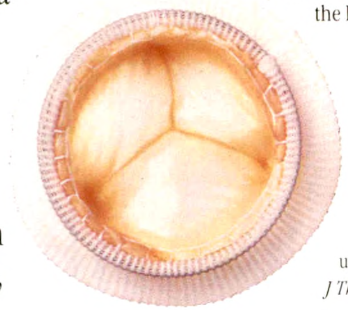

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1. DiSesa VJ, Allred EN, Kowalker W, et al: Performance of a fabricated trileaflet porcine bioprosthesis: Midterm follow-up of the Hancock modified-orifice valve. *J Thorac Cardiovasc Surg* 1987;94:220-224.

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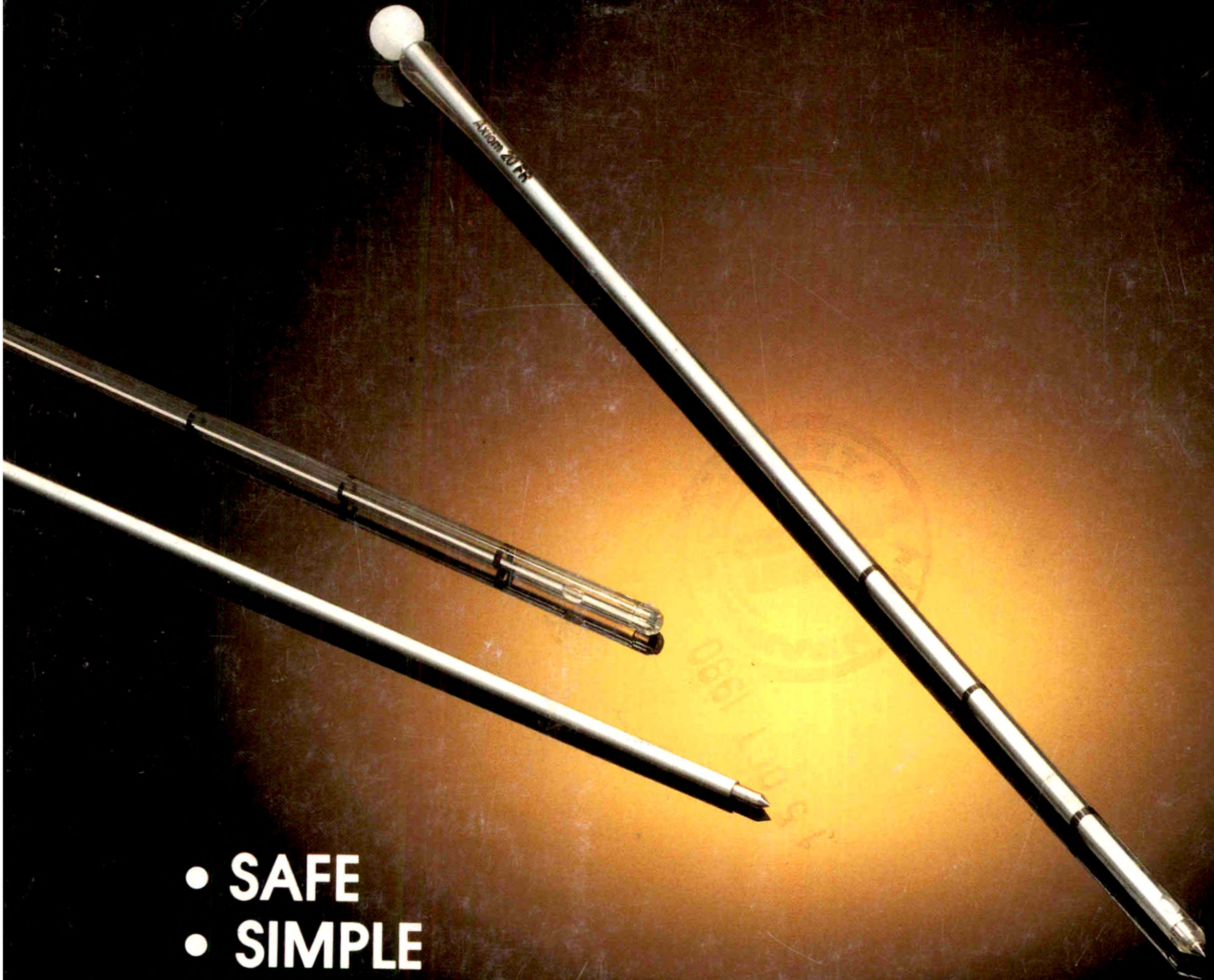

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THE ANNALS OF THORACIC SURGERY

Official Journal of The Society of Thoracic Surgeons
and the Southern Thoracic Surgical Association

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February 18–20, 1991, San Francisco Moscone Center, San Francisco, California
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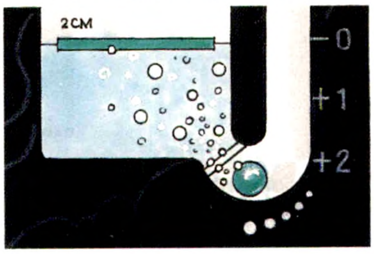

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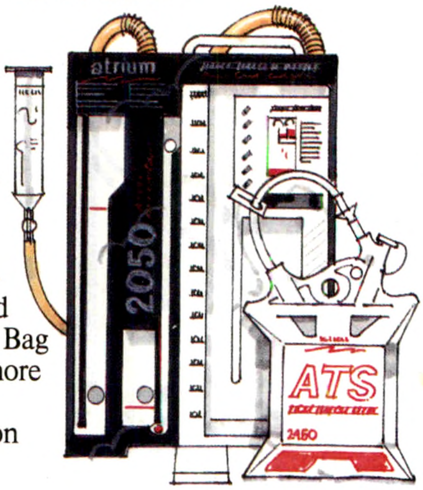

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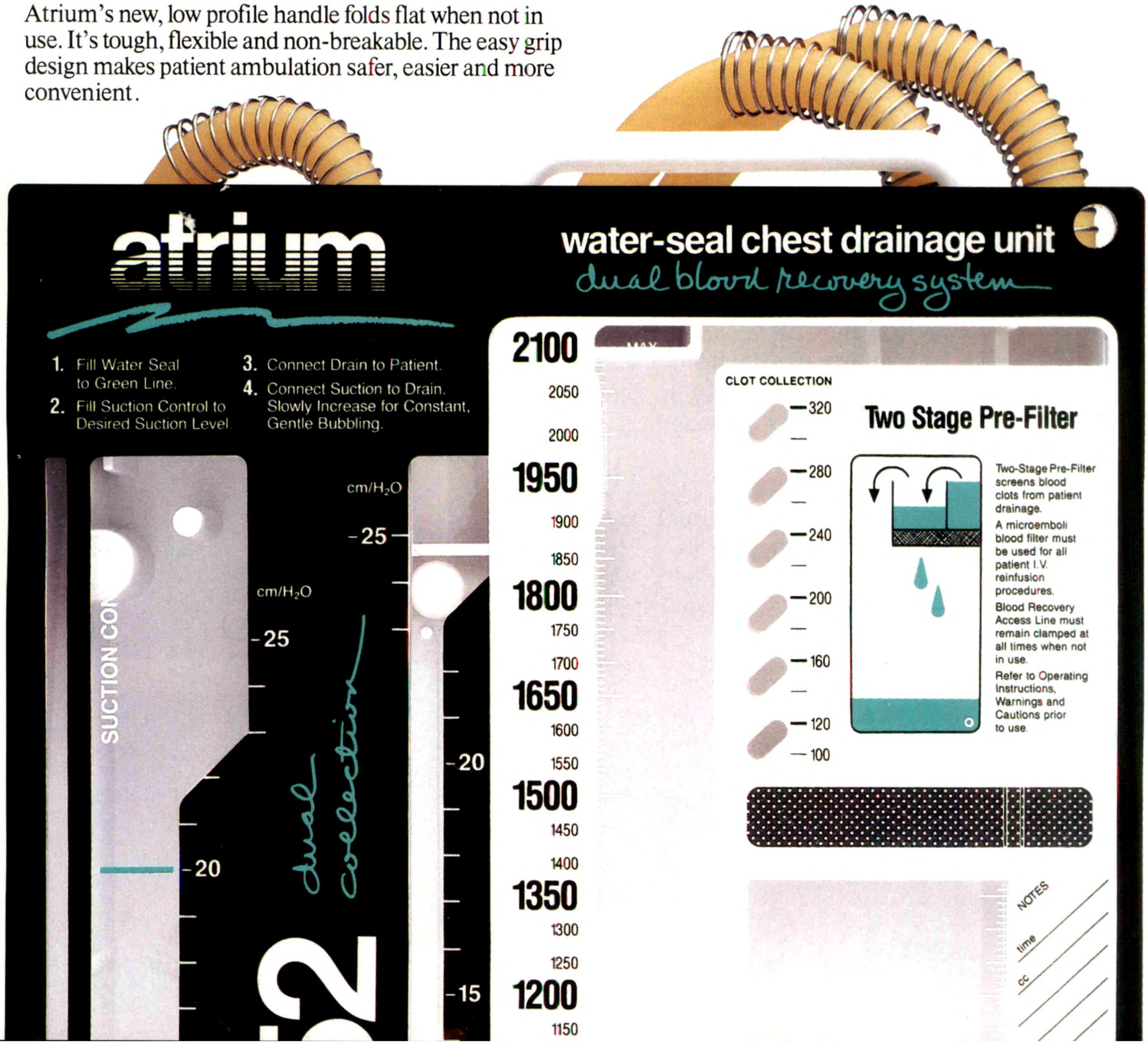
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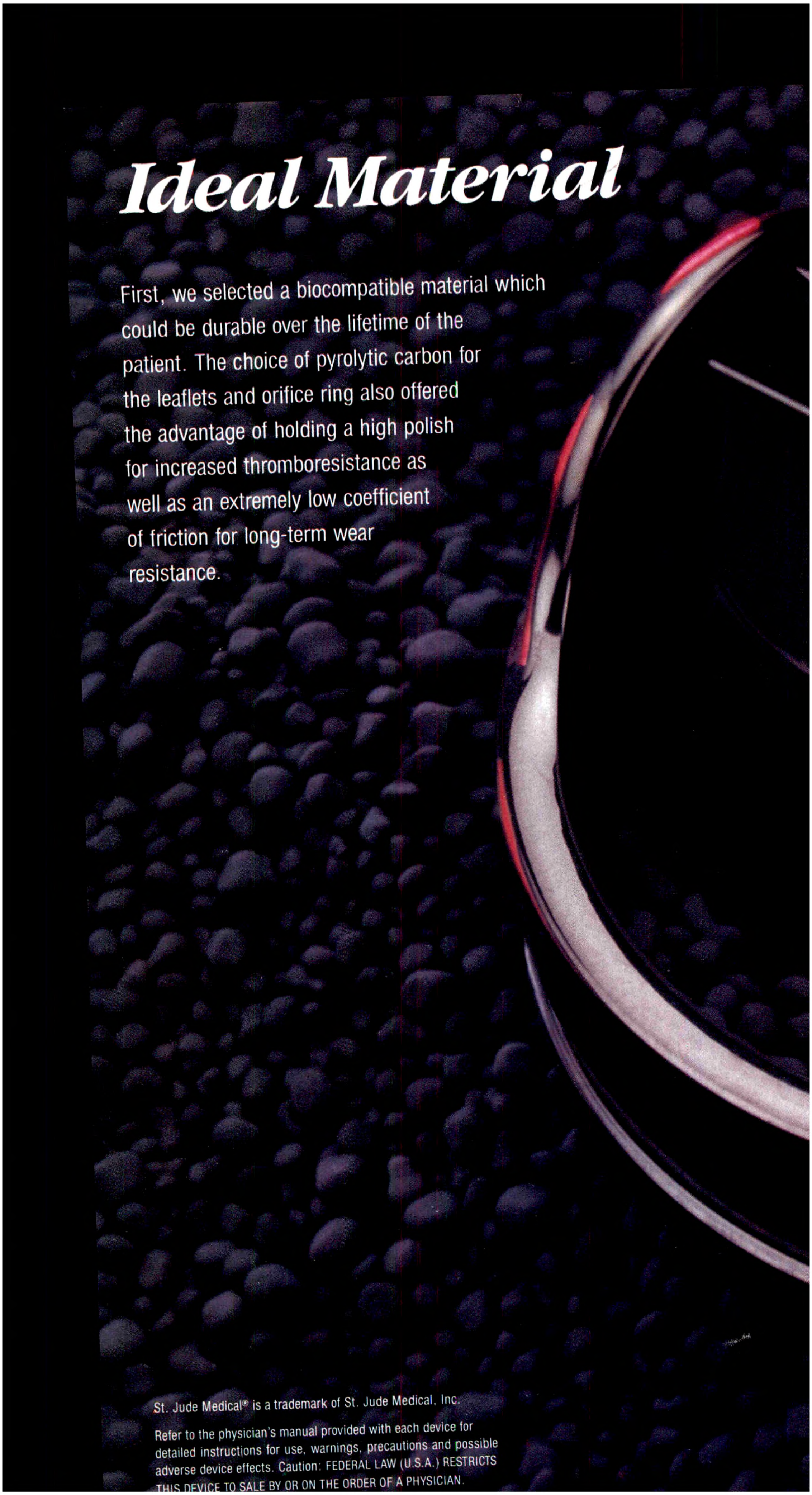

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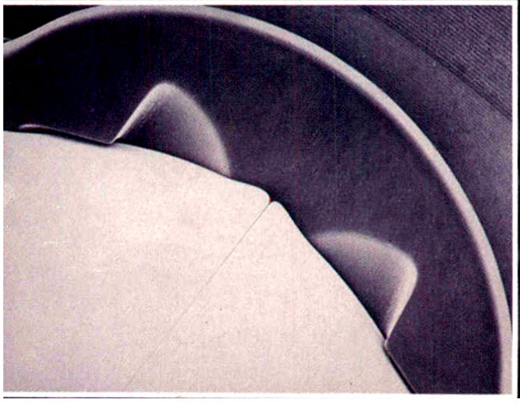

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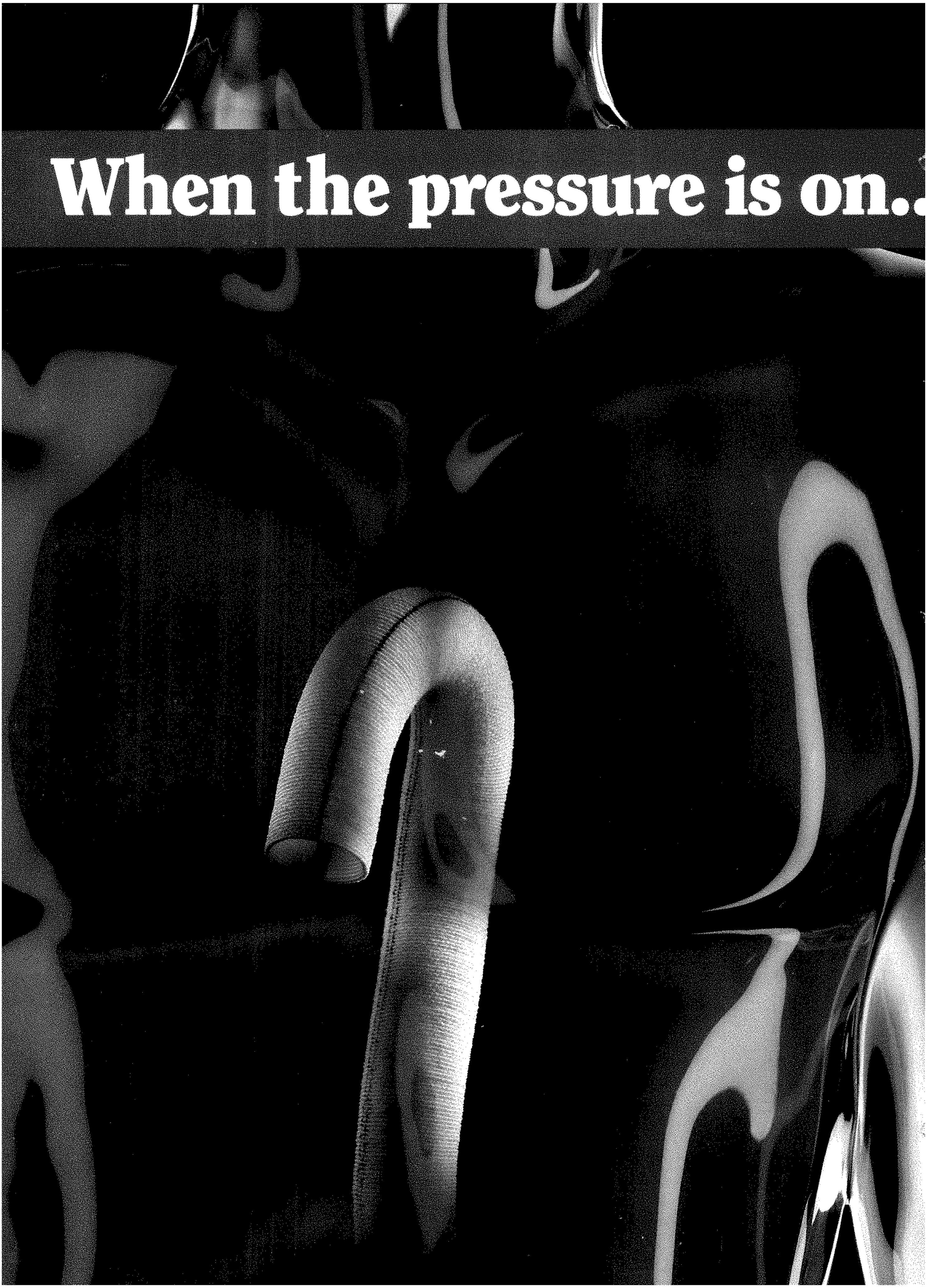
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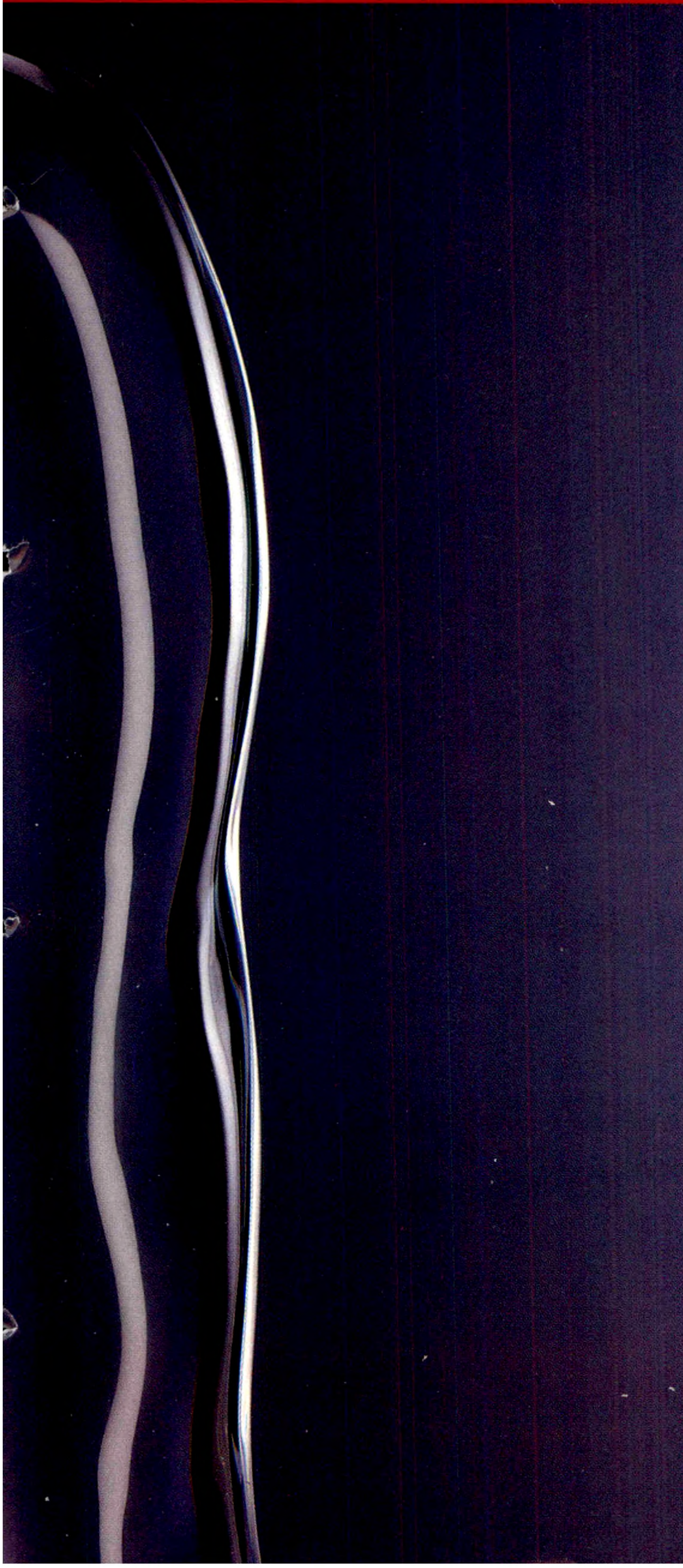

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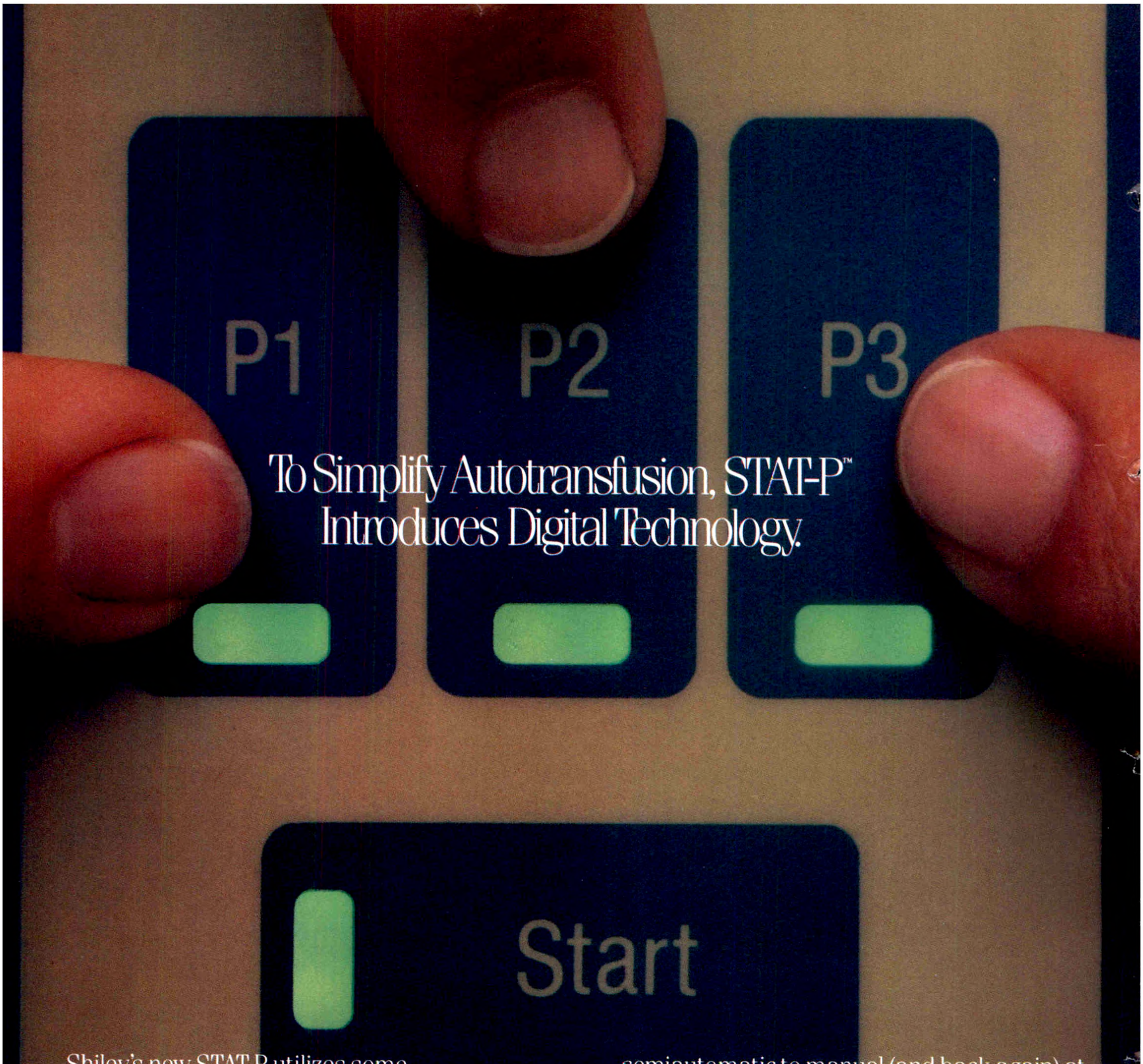

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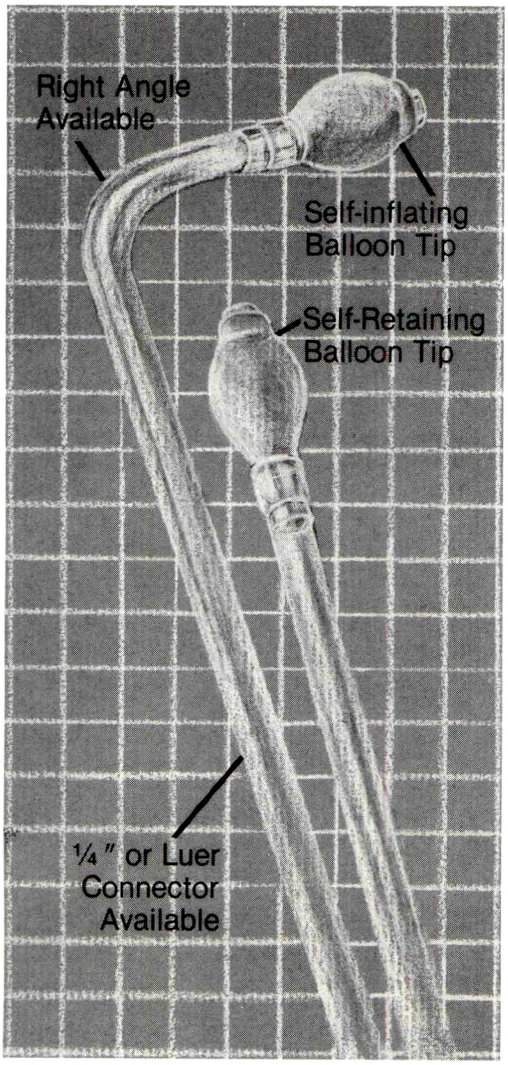
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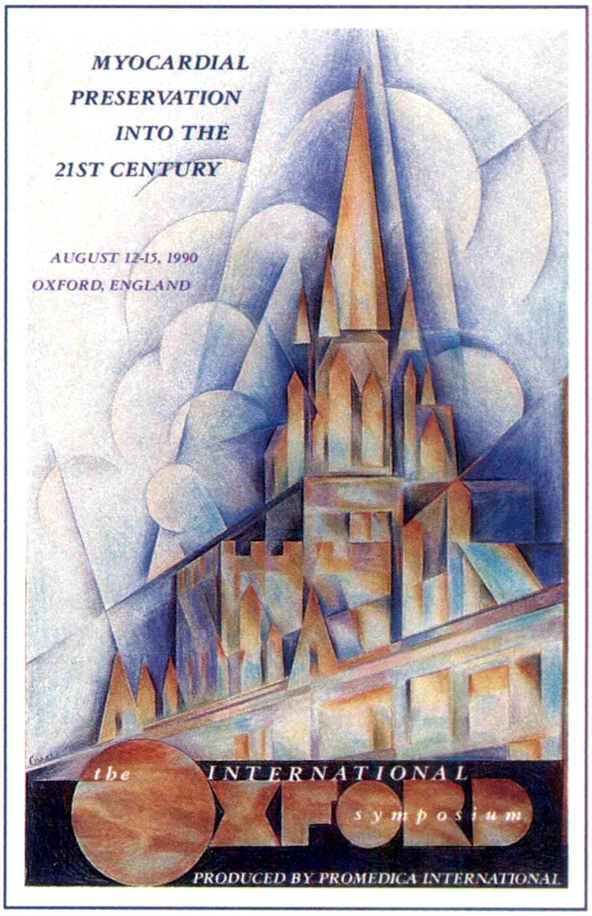
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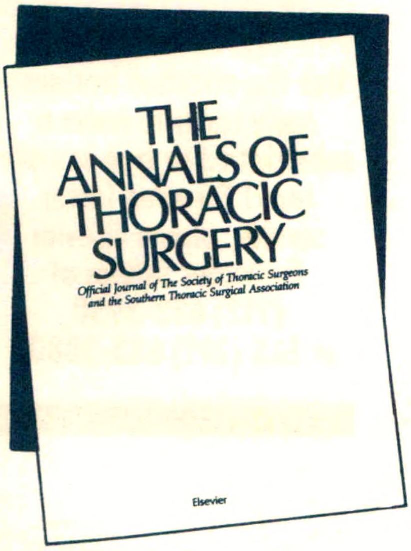
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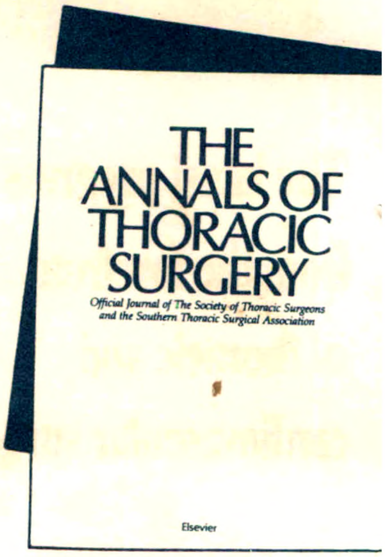

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3. IS-1 is the designation of the standard pacing connector developed by the International Committee on Cardiovascular Implants for Surgery (International Standard Organization ISO TC150/SC).

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Mitral Valve Replacement: What Should Be the Standard Technique?

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Operative mortality reported with mitral valve replacement (MVR) is usually higher than that for aortic valve or coronary bypass procedures. Physiological and chronological age, type of valvar lesion, chronicity, and left ventricular functional deterioration play a role in determining operative and late risks but are immutable at the time of surgical referral. Correction of mitral regurgitation by leaflet and chordal excision and MVR results in decreased total stroke volume because of elimination of low-resistance ventricular decompression into the left atrium. Other pathophysiological features of MVR include substitution of a rigid prosthetic ring for the flexible natural annulus and the potential for myocardial damage during cardiopulmonary bypass and aortic cross-clamping. Increased MVR operative mortality has been noted in patients with mitral regurgitation especially when associated with reduced ventricular function and in patients with mitral stenosis related to the development of posterior transverse left ventricular disruption. Perivalvar leak and low cardiac output states after cardiopulmonary bypass are also associated with increased risk.

Preservation of the continuity of one or both papillary muscles and chordae tendineae with the atrioventricular ring (mitral loop) was originally reported by Lillehei and co-authors [1] to improve cardiac output and reduce operative mortality after MVR. These findings have been generally substantiated [2–5] including in the highest risk patients with acute ischemic mitral regurgitation and markedly reduced ejection fractions (EFs). Shortening of the left ventricular longitudinal axis and circumferential expansion, the response to volume loading, the end-systolic volume index, and the EF at rest and in response to exercise are best maintained or improved when the mitral loop is preserved. Further, few functional differences are demonstrable between patients undergoing repair versus patients undergoing replacement for mitral regurgitation when annular–chordal continuity is maintained [3]. Preservation of the posterior or mural mitral leaflet also appears to largely eliminate posterior transverse left ventricular disruption [6] and may reduce the incidence of perivalvar leak by buttressing the naturally occurring posterior defect in the fibrous mitral annulus.

See also page 866.

The study of Harpole and co-workers [7], which fails to confirm that partial preservation of the mitral loop helps to maintain ventricular function after MVR, raises several concerns. It is small, involving 12 patients, only 8 of whom have mitral regurgitation in which the relevance of chordal preservation to ventricular function is most critical. Even the 8 patients with mitral regurgitation were not treated homogenously, as 1 patient underwent total mitral valve excision and 7 patients had posterior leaflets preserved. Compared with preoperative values, heart rate averaged 8 to 10 beats/min faster during the early and subsequent postoperative studies. Could the potential inotropic effect of increased heart rate have partially masked the effect of mitral loop division? This variable could have been controlled by preoperative and postoperative atrial pacing or by ventricular pacing in patients with atrial fibrillation. Harpole and co-workers define standard MVR as complete excision of the anterior leaflet and preservation of the mural leaflet when convenient; however, they totally excised both the anterior and mural leaflets and chordae in 42% (5/12) of their patients. Thus, posterior leaflet preservation can hardly be defined as standard in their practice, and a review of the recent literature confirms that leaflet and subvalvar apparatus excision preparatory to MVR is still most commonly practiced, especially when a mechanical valve is to be used.

Harpole and co-workers' measures of left ventricular function are independent of numerous physiological variables, but are they of more clinical relevance than studies of left ventricular peak volume elastance and EF indicating that the mitral loop may contribute as much as 40% of maximal left ventricular performance? Although left ventricular wall volume and the relationship of stroke work to end-diastolic volume did not deteriorate after MVR for mitral regurgitation in the present study, several potential clinically relevant variables changed. Both the peak left ventricular pressure and end-systolic wall stress increased while left ventricular EF decreased. Ejection fraction as an index of myocardial function has been questioned because it may be affected by preload, afterload, heart rate, and contractility. However, the clinical relevance of EF is supported by the limited potential for functional recovery and survival after MVR in patients with marked preoperative reductions and/or lack of appropriate response to exercise [8]. As in the present study, low-risk patients with normal preoperative EF may tolerate the acute increase in left ventricular afterload and the reduced total stroke volume and fractional shortening after MVR for mitral regurgitation relatively well. The overall effect is still beneficial, ie, improved ventricular efficiency and forward stroke volume even though EF is reduced. But

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are these data reassuring with regard to management of patients with decreased preoperative EF and acute or chronic mitral regurgitation? This latter group of individuals, who have an increased operative risk and a markedly reduced 5-year survival with standard MVR (by which I mean associated with leaflet and subvalvar excision), will provide a more fertile area for investigation of the potential benefits of mitral loop preservation.

Studies of the type conducted by Harpole and coworkers need to be repeated with a larger number of patients and with a clear-cut separation of treatment subgroups into (1) total valvar excision, (2) total valvar preservation, and (3) posterior apparatus preservation. In each treatment group, patients with mitral stenosis and mitral regurgitation and patients with normal and subnormal ventricular function should be analyzed separately. However, would a study of planned excision of the posterior leaflet be ethical considering all of the data showing a significant reduction in operative risk from posterior transverse left ventricular disruption when the mural leaflet is retained? Because of Lillehei's original report, I have routinely preserved the posterior leaflet since I began practice 20 years ago and coincidentally have never had a patient with posterior transverse left ventricular disruption. Further, essentially all of the publications on left ventricular rupture complicating MVR that detail operative technique report posterior leaflet excision.

A more appropriate question may relate to selective preservation of the anterior leaflet support apparatus. The potential complication of left ventricular outflow tract obstruction is easily avoided by subannular prosthetic positioning and/or rolling up the anterior leaflet like a Roman shade. However, I use interrupted sutures rather than Lillehei's reefing stitch [1], which has a tendency to pursestring the annulus. Partially excising the anterior leaflet [1, 3, 4], also originally described by Lillehei, extends the number of patients in whom the anterior subvalvar apparatus can be preserved. With appropriate valvar sizing and excision of all loose tissues, continued use of mitral loop-preserving techniques with prosthetic mitral valves is supported by my own experience, personal communications, and the literature [1, 4, 5]. Based on the available data, it is probable that many technical and functional problems associated with increased MVR mortality can be ameliorated by preservation of as much of the mitral valve annular and subvalvar apparatus as technically feasible at the time of operation.

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Coarctation of the Aorta: A Surgical Option Revisited

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Resection with end-to-end anastomosis was the initial operation used to correct coarctation of the aorta, but the observed residual gradients and findings of early restenosis in infants led to the development of other surgical procedures. This has been the stimulus for the development of alternative aortoplasty techniques over the past 40 years, including the one reintroduced by Heikkenen and Ala-Kulju in this issue [1].

Vossschulte [2], in 1961, described two techniques applied to a series of 14 patients. A "direct isthmusplasty" of the type described again by Heikkenen and Ala-Kulju was used to alleviate short-segment coarctation of the aorta in 7 patients. Vossschulte also used a patch-repair technique after a longitudinal incision of the descending thoracic aorta from the left subclavian artery to a level beyond the coarctation in 7 patients, and he called this "indirect isthmusplasty." A homograft patch was used for the initial 3 patients in the series, and a Dacron patch was used for the later 4 patients. Some points made in Vossschulte's article included the observation that technical problems in dealing with coarctation arose mainly when the stenosis was long, and that an important principle in repair was the avoidance of a circumferential continuous suture line. Vossschulte believed that the two techniques that he described would best resolve all cases of coarctation while providing for the growth requirements of young patients.

See also page 948.

There seems to be a continuing interest in matching this congenital anomaly with a single "best" surgical procedure. However, the surgical alternatives in addressing coarctation of the aorta are numerous and include direct repair techniques as well as the use of supplemental autologous tissue and synthetic materials. Prosthetic materials have been applied creatively for aortoplasty as patches, gussets, bypass grafts, and interpositions. When relatively noncompliant substitute material is used for the partial replacement of the aortic circumference in the form of a patch, however, there has been an alarming incidence of aneurysm formation involving the remaining aortic wall. It is ironic that Vossschulte's initial patch substitute, a homograft, was probably a better choice. Excision of the coarctation and its replacement with a prosthetic tube remains an excellent technique in long coarctations when further growth of the aorta is not a consideration. The subclavian flap aortoplasty has been shown to be effective and safe, with a low operative mortality and excellent growth potential even in the youngest patients [3]. It is in this group of neonates and infants that the operation has been a substantial success. Ductal tissue found in the wall of the aorta in the region of the coarctation has led some surgeons to believe that the incomplete removal of this abnormal tissue is responsible for recoarctation and has prompted a renewed interest in recent years in more aggressive resection and primary anastomosis. This position was recently reviewed by van Son and colleagues [4].

In dealing with coarctation of the aorta, particularly over a wide age spectrum, the experienced surgeon realizes that there is value in having a host of different techniques available. The contribution of Heikkinen and Ala-Kulju serves to revive our interest in one of these special techniques.

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Tracheal Replacement

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The evolution of tracheal reconstructive surgery in the last 25 to 30 years now permits resection of most tracheal lesions, inflammatory or neoplastic, with primary reconstruction. In general, this may be done safely after resection of up to one half of the trachea in adults and one third in infants and small children. These very broad limits vary widely, depending on age, body build, local anatomy, pathology, and prior therapeutic attempts. In patients with very extensive inflammatory lesions, excision of which would not leave enough native airway for primary reconstruction despite the use of all available adjunctive methods, it is generally possible to open the airway channel by endoscopic or surgical methods sufficiently to place a tailored Montgomery silicone T tube, which provides internal stenting. Although this method does not meet the ideal of reconstruction with native tissues, it provides excellent function with a great margin of safety. The silicone airway thus fashioned is at least as good as any prosthetic device yet used for surgical tracheal replacement. In an effort to make such an airway even more acceptable, surgeons have cut the T arm short and placed it subcutaneously. This, however, eliminates instant access to the airway.

Apart from the desired ideal in treating inflammatory disease of being able to replace the trachea when it cannot be reconstructed with native tissues, the remaining field for tracheal replacement lies with the very small number of malignant tumors that involve a great linear extent of the trachea but that have not invaded the larynx deeply. Such tumors are primarily adenoidcystic carcinomas, in which the diagnosis has been made late or definitive treatment has been delayed by prolonged palliative treatment such as laser therapy. A smaller number of squamous cell carcinomas may also extend over a long length of trachea without extensive mediastinal invasion, which would preclude operation. Even in a clinic such as ours, where a relatively large number of tracheal reconstructions are done annually, only 1 or 2 such patients may be seen in a year. At present we have recommended definitive irradiation as palliation for such lesions, given the presently experimental nature of extended tracheal replacement.

See also pages 951 and 955.

The ideal of replacement, however, remains. It is a particularly intriguing concept, because on the surface it seems so simple to replace a conduit intended primarily for the passage of gases to and from the lung. It has long been demonstrated that secretions can be cleared across tubes of foreign material placed in the trachea and across a tracheal lumen in which the lining has been entirely replaced by squamous metaplasia or by a cutaneous tube. No effort will be made here to cover in any encyclopedic fashion the numerous efforts in tracheal replacement, thus far without a safely acceptable solution. These efforts fall into three general categories: prosthetic replacement, homograft replacement, and autogenous tissue reconstruction. Combinations of these methods abound in the literature.

The fundamental problem encountered with foreign material tracheal replacements has been that ultimately the foreign material lies, after resection of trachea, in a connective tissue bed. As at either end of this bed, epithelium is in contact with an inlying foreign body, a chronic, bacterially contaminated ulcer bed must be accepted as part of the reconstruction. This has led over the years to the twin problems of granulation tissue and chronic infection, which in turn have led to obstruction of the airway, migration of the prosthesis, and erosion of major blood vessels, usually with fatal hemorrhage. This is particularly the case where subtotal tracheal resection is done and a very long replacement is inserted. Shorter replacements are more likely to escape these vicissitudes. The enthusiast must be reminded that short replacements are not necessary, as such cases may be better handled by primary reconstruction. The very limited occasions for such resections have been already noted.

In an effort to solve some of these problems with prostheses, at least two approaches have been tried. The first was the use of an intussusceptive process in which the ends of the foreign tube were slipped into the epithelial-lined tracheal ends, and an outer cuff was sutured tentatively to the outside of the trachea until the inflammation and cicatrization encased the prosthesis. Although some successes have been reported, most experiences with such prostheses have been poor. The second approach has been the use of mesh prostheses of various materials that would permit ingrowth of connective tissue that, in turn, would be epithelialized by migration of tracheal epithelium (with or without additional strip grafts) from either end of the prosthetic tube. The fundamental cause of failure here is that epithelium generally does not migrate more than a short distance from the native trachea, leaving a mass of granulation tissue in the center of the long prosthesis, even assuming that granulations have evenly covered the foreign material. Chronic sepsis has more often resulted.

Experimentation with homografts has a prolonged history. It was discovered early that even the native trachea replaced autogenously immediately after surgical removal often failed to heal appropriately, became necrotic, and ultimately stenosed in an unpredictable number of ani-

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mals. Further, no vascular pedicle can reasonably be obtained to provide revascularization of such a graft by direct microvascular suture. Recently, homografts wrapped in omentum have been tried experimentally with initially enthusiastic reports. An additional factor to be considered in such replacements, of course, is that of immunosuppression.

The third general method of replacement is the use of autogenous tissues in various combinations. Such replacements are too numerous to list. They have included, for example, the creation of skin troughs to connect the larynx above with the supracarinal trachea below, removing the sternum and dropping the surface skin into the depths of the mediastinum if necessary. In staged procedures, various types of splinting prosthetic or autogenous material have been used; they are usually placed in the mesenchyme, without communication to the bacterially contaminated surface. Tubes have also been created at a distance using such combinations as costochondral perichondrium wrapped around a stent, later epithelialized with buccal mucosa or skin grafts, with the whole pedicled on an intercostal muscle to permit its later transfer into a tracheal defect. My colleagues and I used the first technique (cutaneous tubes) clinically and the second technique (perichondrial tubes) experimentally. Both suffered the usual result of such complex techniques—a few successes, but far too many failures to justify clinical use.

The use of intestinal tubes, esophageal or jejunal (the latter on a revascularized pedicle), seems attractive, and has been tried by a number of investigators. Two such approaches are described in this issue. The first approach is that of Kato and colleagues [1]—interposition of the local esophagus into a gap where the trachea has been resected. The interposition simply provides a bed into which a silicone T tube is placed, avoiding the problems of the chronic ulcerated bed that results when prosthetic material alone is used. As such a replacement would be used only in the case of extended or subtotal tracheal resection, the esophagus would necessarily have to be reconstructed either with stomach, colon, or vascular-pedicled jejunum because in humans the gap would be too long for end-to-end reconstruction. If end-to-end anastomosis of the esophagus were possible, then the tracheal resection would have been short enough to permit its end-to-end anastomosis.

Although the ingenuity of surgeons now provides a number of mechanisms for replacement of the esophagus after resection, it is well recognized that none of these function in a manner resembling normal esophagus. All such replacements, especially over the long term, continue to present the patient with multiple difficulties of function. Although a patient may be willing to accept this where esophagectomy has been necessary because of intrinsic esophageal disease, I find the exchange of normal esophageal function for T tube establishment of the airway to be somewhat dubious. Clearly, this would be justified if otherwise lethal disease is being resected, but such massive surgical alternatives should be approached circumspectly at best. I should point out that attempts to stabilize similar esophageal replacement to provide a "tracheal" wall without a T tube have succeeded only episodically in the past. Some years ago, I attempted such experiments using polypropylene rings, but valving with airway obstruction was a serious problem.

Letang and associates [2] used another route—replacement of the resected tracheal segment with free revascularized small intestinal graft. In this series, the graft did not have to be used as a conduit for the placement of a stenting T tube, but achieved rigidity on its own and permitted the experimental animals to respire essentially normally. This is difficult to explain biologically, even with the inflammation that would normally bind the wall of the transplanted segment to the surrounding mediastinal tissues. With softening of this inflammatory reaction over time, one might expect that the airway would not remain patent, especially if the segment were long. There may be a factor of relative diameters, given the large diameter of the dog's trachea and relatively small diameter of its intestine, with the reverse relationship being present in humans. Once again, it is a method of sufficient complexity that its clinical application would best be pursued with caution.

We must continue to maintain an open mind about this intriguing but thus far unsolved surgical dilemma—replacement of the tracheal conduit. Surgical ingenuity will continue to attack these problems in a host of different ways. The investigator should be closely guided in his or her concepts by the realistic reparative tissue responses. The clinical surgeon must be cautioned against rushing to apply experimental concepts to a single case. Innovative replacements would best be pursued in a systematic fashion by a team that is exploring a particular avenue. Needed also are candid and unvarnished reports of attempts at replacement that have been carried out clinically in a controlled and considered manner, even where the results are disastrous, as so far they have so often been. The methods of replacement of the trachea must not create more problems than they solve. Surgically acceptable methods must be safe and not just occasionally successful. We must remember that even where a series of animal experiments is successful, application of these techniques to humans almost inevitably presents greater problems and a higher failure rate.

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Effects of Standard Mitral Valve Replacement on Left Ventricular Function

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Recent studies have suggested that excision of the mitral valve apparatus during mitral valve replacement impairs left ventricular performance. However, functional measurements in humans have been difficult to obtain in a load-independent fashion. To investigate this concept, 12 patients (mean age, 65 ± 8 years; mean New York Heart Association functional class, 3.3 ± 0.7) with 4+ mitral regurgitation (n = 8) or mitral stenosis (valve area, 1.2 ± 0.2 cm^2) (n = 4) underwent prosthetic valve replacement using crystalloid cardioplegia. No patient required therapeutic inotropic support, every patient had at least the anterior mitral leaflet excised, and paced heart rate was maintained constant throughout. Left ventricular volume was measured with radionuclide angiocardiography, left ventricular pressure with a 3F micromanometer, and left ventricular wall volume with two-dimensional transesophageal echocardiography. Left ventricular preload was varied over a mean end-diastolic pressure range of 9 to 20 mm Hg and an end-diastolic volume range of 134 to 170 mL to generate four to five steady-state pressure–volume loops before and ten minutes after cardiopulmonary bypass. Left ventricular performance was estimated with the stroke work/end-diastolic volume relationship, which is insensitive to load. After bypass, no significant change ($p > 0.1$) was noted in wall volume for patients with mitral regurgitation or mitral stenosis (175 ± 68 to 189 ± 63 mL/m^2 and 130 ± 22 to 127 ± 19 mL/m^2, respectively). The stroke work/end-diastolic volume relationships were highly linear before and after bypass with a mean linear regression coefficient of 0.96 ± 0.03, and the slope and x-intercept were not changed after bypass ($p > 0.1$). These data suggest that partial excision of the mitral apparatus does not significantly impair left ventricular performance during the early period after routine valve replacement.

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Much debate has appeared in the scientific literature over the past 30 years concerning the effects of mitral valve procedures on left ventricular function [1–15]. Retrospective clinical studies predominate, and few data are available to quantify left ventricular function when a change in systolic load occurs. Earlier work [16] from our center described a technique for measuring left ventricular function in a load-independent fashion using serially recorded micromanometric left ventricular pressures and initial-transit radionuclide angiocardiographic (RNA) volumes before and immediately after cardiopulmonary bypass. The purpose of this project was to evaluate ventricular performance in humans during mitral valve replacement and to test the hypothesis that partial or complete resection of the native mitral valve produces an acute decrement in left ventricular function.

For editorial comment, see page 861.

Material and Methods

Population

This study was approved by the Duke University Institutional Review Board, and written informed consent was obtained from all participants. The population consisted of 12 patients with hemodynamically documented 4+ mitral regurgitation (MR) (n = 8) or severe mitral stenosis (MS) (valve area, 1.2 ± 0.2 cm^2) (n = 4). There were 5 men and 7 women with a mean age of 65 ± 8 years. Symptoms of New York Heart Association class III or IV congestive heart failure were present in all. No patient had major coronary artery disease, a prior myocardial infarction, or a previous cardiac operation. The valve anatomy in each patient was judged intraoperatively to be unsuitable for valve repair.

Experimental Design

After the induction of anesthesia with intravenous administration of fentanyl and midazolam hydrochloride, endotracheal intubation was accomplished. The cardiopulmonary bypass circuit consisted of standard aortic and bicaval venous cannulation for all mitral valve operations. A 3F high-fidelity micromanometer (Millar Instruments, Inc, Houston, TX) was calibrated and inserted into the left ventricle through a transseptal approach. A Scinticor gamma camera (Scinticor, Inc, Milwaukee, WI) enclosed

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in a sterile sheath was positioned over the heart, and four to five initial-transit radionuclide angiocardiograms were acquired during steady-state periods after the addition or withdrawal of blood volume from the aortic cannula. During acquisition of RNA data, two-dimensional transesophageal echocardiograms (Hewlett-Packard, Inc, Sunnyvale, CA) were also recorded.

Cardiopulmonary bypass was initiated at a calculated flow rate of 2.0 L/min/m^2, and systemic cooling was begun to 24° to 28°C. Topical cooling consisted of iced saline slush, and 700 to 1,200 mL of 4°C potassium crystalloid cardioplegia was infused after aortic cross-clamping to keep myocardial temperature less than 15°C. The term *standard mitral valve replacement* was defined as complete excision of the anterior mitral leaflet in all cases and preservation of the posterior leaflet whenever convenient. Total excision of both mitral valve leaflets and chordae tendineae was accomplished in all patients with MS and in 1 of the 8 patients with MR. Interrupted mattress sutures supported with Teflon felt pledgets were used for all valve insertions.

After completion of the valve procedure, rewarming was begun, and the heart was electrically defibrillated if a spontaneous rhythm did not occur. Epicardial atrial and ventricular pacing electrodes were placed, and pacing was used to maintain a similar heart rate for the second set of studies. Cardiopulmonary bypass was discontinued after rewarming to a rectal temperature of 36°C; ten minutes after discontinuation of cardiopulmonary bypass, the RNA and echocardiographic studies were repeated. Twenty-four hours postoperatively, each patient had a repeat RNA study in the intensive care unit after endotracheal extubation and discontinuation of intravenous medications.

Radionuclide Angiocardiography

Initial-transit radionuclide angiocardiograms were acquired at 20-ms intervals using the Scinticor multicrystal gamma camera. Ten millicuries of technetium 99m–labeled DTPA (diethylenetriamine pentaacetic acid) was injected as a bolus through an internal jugular venous Teflon catheter with a data acquisition time of 15 to 20 seconds for each study. Scinticor software was used to calculate heart rate, cardiac output, left ventricular ejection fraction, left ventricular end-diastolic volume, end-systolic volume, and peak ejection rate. The accuracy and reproducibility of these measurements have been reported in previous investigations [17–19]. High-fidelity left ventricular pressure data were recorded with each 20-ms radionuclide image, thus allowing construction of pressure–volume loops from an average cardiac cycle using a method validated by previous work from our laboratory [20].

Two-Dimensional Echocardiography

Two-dimensional Doppler echocardiograms were obtained using a 5-MHz transesophageal Hewlett-Packard echocardiographic probe. Projections recorded on videotape included left ventricular long axis and left ventricular mid-papillary muscle short axis. Data were analyzed for the estimation of left ventricular (LV) wall volume using an area–length relationship: Wall volume = [(LV epicardial short-axis area × LV epicardial long-axis dimension) − (LV endocardial short-axis area × LV endocardial long-axis dimension)] [21]. Left ventricular end-systolic meridional wall stress (10^3 dynes/cm^2) was estimated for each patient before and after the valve operation with the equation: Wall stress = [0.334 × LV end-systolic dimension (ESD) × LV end-systolic pressure]/[end-systolic wall thickness (h) × (1 + h/ESD)] [22, 23].

Left Ventricular Performance

To evaluate ventricular performance, serial pressure–volume loops obtained before and after each valve replacement were constructed, and the integrated loop area, or left ventricular stroke work, was obtained. The preload recruitable stroke work model assesses the slope and x-intercept of the linear regression of stroke work versus left ventricular end-diastolic volume [24]. If a significant perioperative change is observed in linear regression coefficients, a change in left ventricular performance is assumed [16]. In addition, the end-systolic pressure–volume coordinates for each RNA loop were plotted before and after the procedure to assess ventricular performance using the Suga-Sagawa relationship [25, 26].

Statistical Analysis

Significance of change was estimated for each variable measured before and after the valve procedures using Student's paired *t* test and analysis of variance with a level of significance at 0.05. Analysis of covariance was used to assess changes in linear regression coefficients.

Results

All patients tolerated the surgical procedure well, and no perioperative complications occurred. Cardiopulmonary bypass and aortic cross-clamp times were not significantly different for the two valvular lesions (Table 1). The mean preload range obtained for the four or five RNA studies before and after the valve procedure was 9 to 20 mm Hg for left ventricular end-diastolic pressure and 134 to 170 mL for left ventricular end-diastolic volume (see Table 1). Heart rhythm was normal sinus for 10 patients before the procedure and atrial fibrillation in 2 patients with MS; afterwards, the rhythm was normal sinus in 3 and atrially paced in 7. The 2 patients in atrial fibrillation were ventricularly paced at a regular rate after valve replacement. Average heart rate increased slightly from 81 ± 14 before operation to 89 ± 7 beats per minute after the procedure (p = not significant). No patient required therapeutic inotropic support, defined as 4 μg/kg/min or more of dopamine hydrochloride, and inhaled anesthetic agents or other contractile-depressing medications were avoided during the study period.

Using the area–length method, left ventricular wall volume, calculated from the echocardiographic images, did not change postoperatively (MR, 175 ± 68 preoperatively to 189 ± 63 mL/m^2 postoperatively; MS, 130 ± 22 to 127 ± 19 mL/m^2) (Fig 1). Left ventricular end-systolic meridional wall stress did change significantly for the

Table 1. Cardiopulmonary Bypass Times and Preload Ranges

Variable	Mitral Regurgitation	Mitral Stenosis
Cardiopulmonary bypass (min)[a]	98 ± 27	92 ± 30
Aortic cross-clamping (min)[a]	55 ± 18	54 ± 22
Preload range preop		
LV end-diastolic pressure (mm Hg)	12–21	7–15
LV end-diastolic volume (mL)	156–193	89–131
Preload range postop		
LV end-diastolic pressure (mm Hg)	11–23	7–19
LV end-diastolic volume (mL)	150–205	97–142

[a] Data are shown as the mean ± the standard deviation.

LV = left ventricular.

patients with MR (32.6 ± 24.0 to 57.8 ± 40.1 × 10^3 dynes/cm^2; $p < 0.01$), but no change in afterload was noted for the 4 patients with MS (21.3 ± 4.3 to 20.7 ± 7.9 × 10^3 dynes/cm^2) (Figs 2, 3). Mean left ventricular ejection fraction ranged from 0.48 ± 0.09 preoperatively to 0.37 ± 0.11 postoperatively for patients with MR ($p < 0.01$), but was unchanged for patients with MS (0.51 ± 0.10 to 0.52 ± 0.12). Peak left ventricular pressure increased in MR patients (82 ± 11 to 109 ± 20 mm Hg; $p < 0.001$); an insignificant change was observed in MS patients (81 ± 13 to 89 ± 13 mm Hg). Studies acquired 24 hours after operation revealed no change in heart rate (89 ± 8 to 91 ± 9 beats per minute), and left ventricular ejection fraction measured immediately after the valve procedure was maintained one day postoperatively for all patients (Fig 4).

Stroke work/left ventricular end-diastolic volume relationships before and after valve replacement were highly linear with mean linear regression coefficients of 0.96 ± 0.03 and 0.98 ± 0.02, respectively (Figs 5, 6). Variation in the stroke work/end-diastolic volume relationship before and after valve operation was measured using an analysis of covariance, and no significant change was noted for the slope (8.4 ± 2.6 to 8.0 ± 2.9 10^4 ergs/mL) or x-intercept (75 ± 32 to 61 ± 37 mL) (Tables 2, 3; Fig 7). In addition, no significant perioperative change was observed for the Suga-Sagawa relationship slope (1.0 ± 0.5 to 0.9 ± 0.4 mm Hg/mL) and volume intercept (−27 ± 55 to −24 ± 50 mL) (Fig 8).

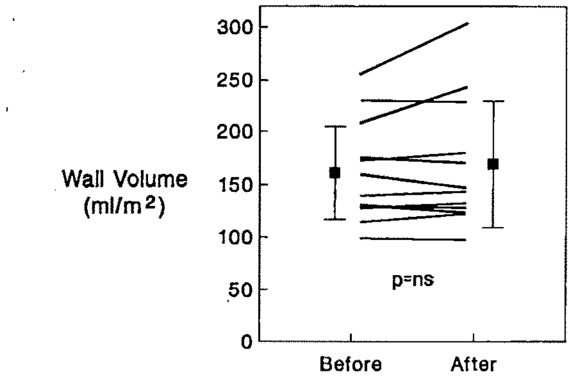

Fig 1. Two-dimensional transesophageal echocardiographic data were used to estimate left ventricular wall volume before and after valve operation using an area–length relationship. No significant (ns) *change was observed after valve operation.* Bars *represent the mean ± the standard deviation.*

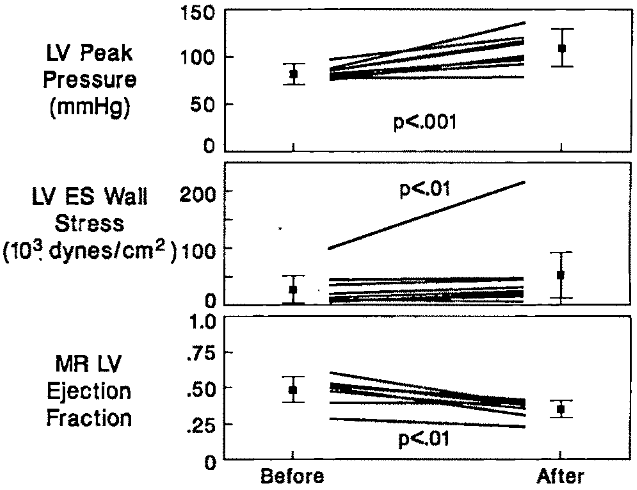

Fig 2. Perioperative changes in peak left ventricular (LV) pressure, end-systolic (ES) meridional wall stress, and ejection fraction are demonstrated for patients with mitral regurgitation (MR). Note that all variables changed significantly after valve replacement. Bars *represent the mean ± the standard deviation.*

Comment

Clinical results obtainable after mitral valve replacement have improved over the last 25 years since the publication

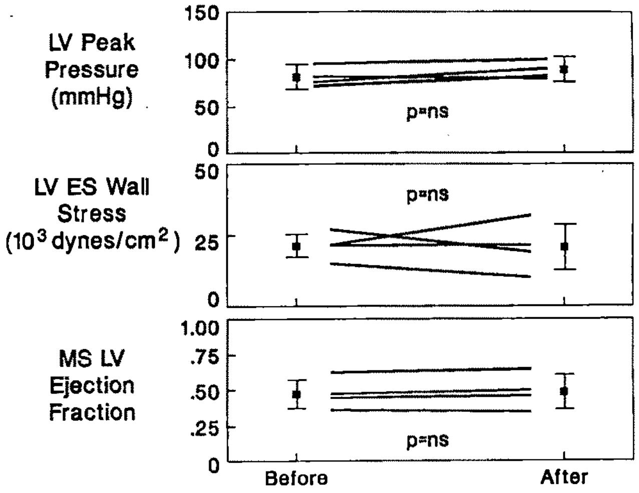

Fig 3. The peak left ventricular (LV) pressure, end-systolic (ES) meridional wall stress, and ejection fraction are demonstrated for patients with mitral stenosis (MS). After valve replacement, no significant (ns) *change was observed for any of the variables.* Bars *represent the mean ± the standard deviation.*

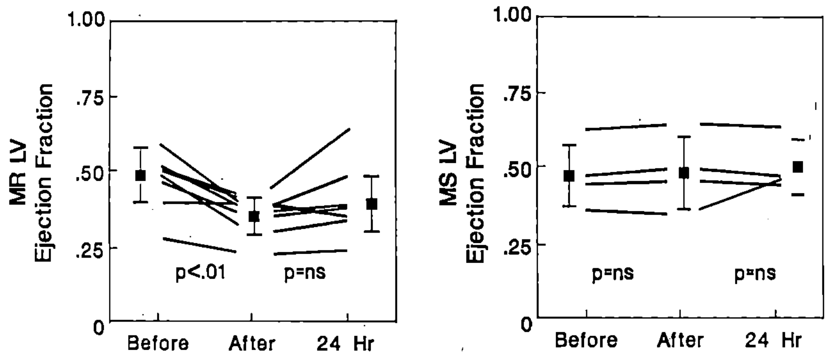

Fig 4. The left ventricular (LV) ejection fraction is demonstrated for the valvular lesions before, immediately after, and 24 hours after operation. No significant (ns) change was observed at 24 hours for patients with either mitral regurgitation (MR) or mitral stenosis (MS). Bars signify the mean ± the standard deviation.

of a technique for preservation of the posterior mitral valve leaflet by Lillehei and associates [1]. Preservation of the mitral valve apparatus was thought to be important for reducing the incidence of posterior myocardial rupture and also for preserving normal geometry and systolic performance of the left ventricle, a concept described by Wiggers and Katz [5] in 1922 and Rushmer and associates [9] in 1956.

The technique of Lillehei and co-workers [1] was based on operative data from 13 dogs and, once applied to humans, was associated with a reduction in operative mortality from 37% to 14%. Rastelli and co-workers [2] measured postoperative left ventricular function in dogs undergoing mitral valve replacement with and without preservation of the mitral apparatus. No difference was observed between the two groups; however, ventricular function was quantified with only cardiac output and filling pressures, variables subject to other physiological influences. Spence and associates [27] measured ventricular function during simulated mitral valve replacement with and without preservation of the chordae tendineae and found a significant decrease in peak left ventricular pressure and peak rate of rise of left ventricular pressure after resecting the valve apparatus. These data were obtained on isolated porcine hearts, however, and myocardial function was measured with less than optimal methods.

Hansen and associates [13, 14] used intracavitary balloons in isolated canine left ventricles to measure the effects of severing the chordae tendineae on ventricular function. When the chordae were disconnected, dyskinetic myocardial regions were created and maximal elastance decreased. However, this group used an acute animal model with intracavitary balloons held in place by a plastic ring in the ventricle, and this rigid device could have interfered with contraction. The applicability of the maximal elastance model is also questionable in this setting.

Spratt and co-authors [15] created a short-term chronic left ventricular–left atrial shunt in conscious dogs to simulate chronic mitral regurgitation. During shunt closure, left ventricular wall stress, peak pressure, ventricular efficiency, and forward stroke volume increased, while total stroke volume and fractional shortening decreased. These data confirmed those in previous studies indicating that correction of MR involved an acute increase in left

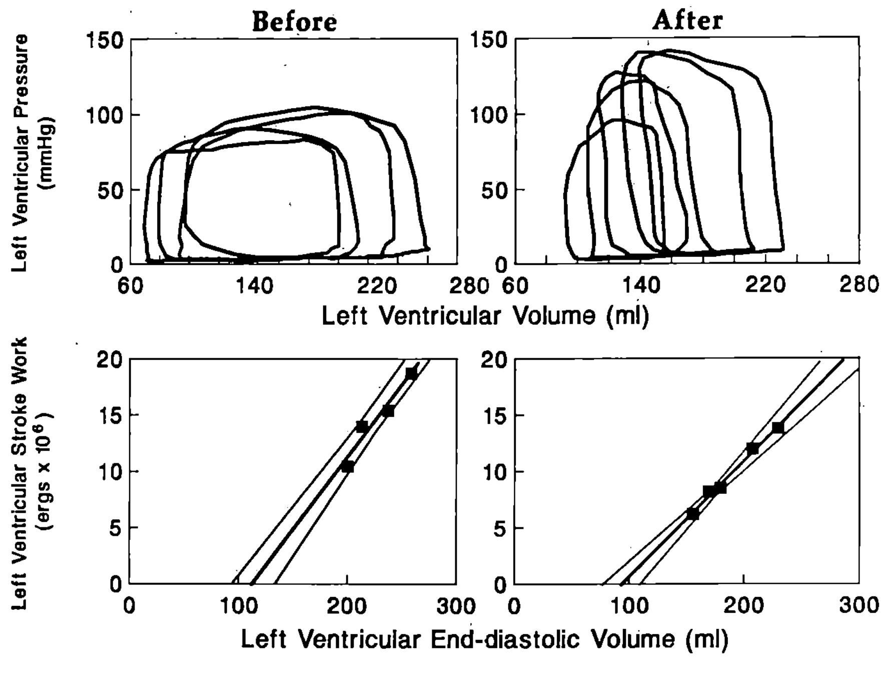

Fig 5. Representative left ventricular pressure–volume loops and stroke work/end-diastolic volume data from a patient with mitral regurgitation demonstrate a significant increase in left ventricular load measured and a decrease in ejection fraction after valve replacement. Despite the marked hemodynamic alteration, stroke work/end-diastolic data were not different.

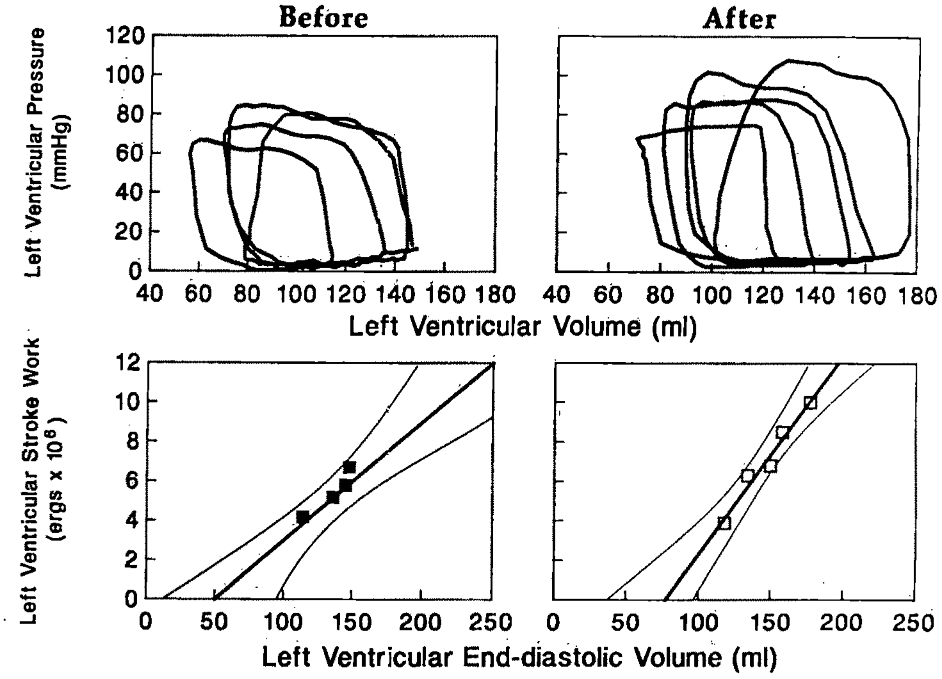

Fig 6. Data for a patient with mitral stenosis are shown demonstrating the significant increase in the left ventricular preload range after operation. However, left ventricular loading, ejection fraction, and stroke work/end-diastolic volume relationships did not change.

ventricular afterload, even when global myocardial performance was relatively unchanged. This increase in afterload uniformally reduced global ejection fraction, even though pump function was improved (in terms of a higher forward cardiac output obtained at a lower filling pressure). Because of the inherent change in systolic loading associated with correction of MR, ejection-phase indices of cardiac performance are less useful.

Retrospective clinical studies evaluating left ventricular function before and after mitral valve replacement have suggested that preservation of the entire mitral apparatus could maintain ventricular function at a more normal level. David described a technique for preservation of the entire mitral apparatus during mitral valve replacement, but recommended its use for bioprostheses only [28, 29]. Operative time was increased by this procedure, and most of the valves may have been amenable to valve repair. David and co-workers [12] also published a retrospective study comparing ventricular function in three groups of patients: mitral valve replacement with resection of the entire mitral apparatus (n = 6), mitral valve replacement with chordal preservation (n = 6), and mitral valve repair (n = 5). Left ventricular ejection fraction fell in group 1, but was maintained in groups 2 and 3. This was a retrospective study with a small sample size, and myocardial function was measured with a load-dependent variable.

In other studies [30], intraoperative echocardiography has defined dyskinesia in the posterior segments of the left ventricle and a decrease in global ejection fraction in

Table 2. Data Range for Pressure–Volume Loops[a]

Variable	Preop	Ten Minutes Postop
Cardiac output (L/min)	5.3 ± 2.0 to 7.4 ± 2.4	4.5 ± 1.2 to 6.5 ± 1.3
LV end-diastolic pressure (mm Hg)	10 ± 4 to 19 ± 4	10 ± 3 to 22 ± 4
LV end-diastolic volume (mL)	134 ± 43 to 172 ± 44	133 ± 39 to 184 ± 41
LV stroke work (10^6 ergs)	4.7 ± 2.2 to 8.2 ± 3.6	5.1 ± 1.8 to 9.3 ± 3.0
LV end-systolic pressure (mm Hg)	67 ± 8 to 85 ± 8	82 ± 15 to 110 ± 20
LV end-systolic volume (mL)	69 ± 22 to 87 ± 24	80 ± 33 to 112 ± 37

[a] Data are shown as the mean ± the standard deviation.

LV = left ventricular.

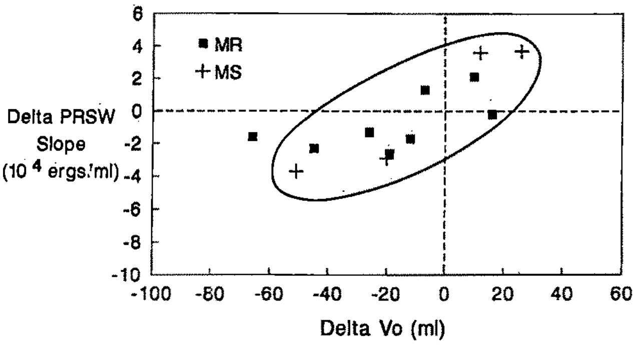

Fig 7. Data for the entire population were compared using an analysis of covariance. No significant change was noted in the slope (preload recruitable stroke work [PRSW]/end-diastolic volume) or the x-intercept (Vo). This is demonstrated with a plot of the 95% confidence ellipse around the difference between the slope and x-intercept after operation. Note that the origin is enclosed in the ellipse, signifying that there was no change in performance after cardiopulmonary bypass. MR = *mitral regurgitation;* MS = *mitral stenosis.*

Table 3. Linear Regression Analysis of Stroke Work/End-Diastolic Volume Relationship

Patient No.	Age (yr)	Disease	R^2		Slope (10^6 ergs/mL)		X-Intercept (mL)	
			Preop	Postop	Preop	Postop	Preop	Postop
1	65	MR	0.99	0.97	5.1	3.5	71	5
2	62	MR	0.99	0.97	10.0	8.7	68	42
3	58	MR	0.98	0.99	3.5	3.3	42	58
4	64	MR	0.93	0.98	10.5	12.6	103	113
5	61	MR	0.90	0.99	12.1	10.4	145	133
6	77	MR	0.95	0.98	8.4	6.1	106	61
7	76	MR	0.95	0.94	7.5	8.8	78	71
8	74	MR	0.97	0.99	12.8	10.2	111	92
9	53	MS	0.99	0.99	9.4	5.7	65	14
10	63	MS	0.99	0.93	8.4	5.5	46	26
11	67	MS	0.94	0.98	6.3	10.0	50	76
12	69	MS	0.98	0.96	7.1	10.7	30	42
Mean[a]	65 ± 8	. . .	0.96 ± 0.03	0.98 ± 0.02	8.4 ± 2.9	8.0 ± 2.9	75 ± 32	61 ± 37

[a] Data are shown ± the standard deviation.

MR = mitral regurgitation; MS = mitral stenosis; R^2 = linear regression coefficient.

patients who had undergone complete excision of the mitral apparatus for MR. However, wall motion, again, is inadequate as the sole measurement of ventricular performance in this setting, and the additional possibility of undefined inadequacies in myocardial protection was not addressed in this, or for that matter most, clinical studies.

This project was designed to more precisely measure left ventricular performance before and early after mitral valve replacement under human pathophysiological conditions using a load-insensitive method [16]. The technique for obtaining serial first-pass RNA pressure–volume loops intraoperatively has been validated in several publications [17–19]. The population of this study consisted of patients with single valvular lesions of either MR or MS and no previous cardiac operations. Major coronary artery disease was not present, and no patient had had a previous myocardial infarction. Mitral regurgitation was uniformly degenerative in origin, and MS was due to rheumatic valvular stenosis. Echocardiographic left ventricular wall thickness and wall volume measurements were significantly elevated for MR patients, consistent with advanced eccentric hypertrophy due to volume overload.

Left ventricular ejection fraction has been used extensively as an index of ventricular function; however, ejection fraction is highly load dependent and varies significantly with changes in left ventricular systolic wall stress [15, 22, 23]. This point is supported in the present study by the decrement in ejection fraction observed early after valve replacement for MR. The absence of significant perioperative variations in systolic loading (wall stress) in patients with MS also correlated well with the stable ejection fractions noted in this disorder. Except for a slightly increased preload range after valve replacement, no significant change in ventricular performance was observed in patients with MS. Because of chronic endocardial scarring involving subvalvular structures in rheumatic MS, our findings reinforced previous clinical studies indicating little ventricular impairment after valve replacement involving mitral excision.

Importantly, however, most of the debate in the literature concerns patients with MR. Other than the increased

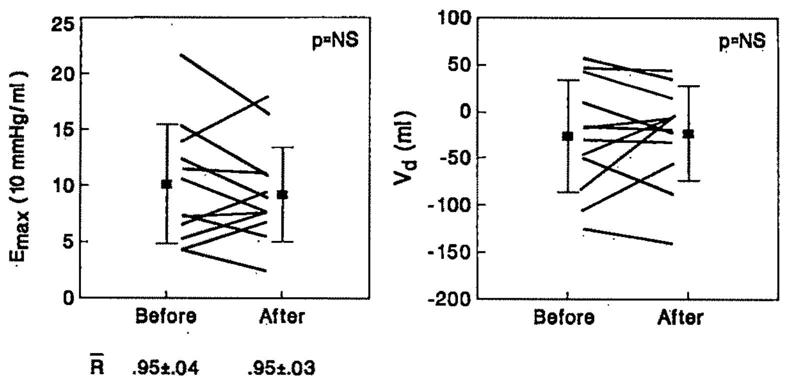

Fig 8. Data for the entire population were compared using Student's paired t *test and an analysis of variance. No significant change* (NS) *was noted in the slope* (E_{max}) *or the x-intercept* (V_d) *of the Suga-Sagawa relationship [25] after valve replacement. The mean linear regression coefficients* ($\bar{R}$) *before and after valve operation also are shown.* Bars *signify the mean ± the standard deviation.*

global afterload observed in those with MR after restoration of valvular competence, no significant change in myocardial performance was observed with standard mitral valve replacement when data from the entire population were examined using load-insensitive stroke work/end-diastolic volume and Suga-Sagawa relationships. This finding may be surprising in view of recent publications to the contrary. However, it must be stressed that our data were obtained in humans under clinical conditions, and given the highly validated nature of the analytical methods, these results have major implications.

Rejecting the hypothesis tested in this study, our results indicate that partial or complete resection of the mitral valve apparatus during prosthetic valve replacement did not seem to impair intrinsic left ventricular function. Perhaps previous conclusions describing a decrement in performance after valve replacement were based on inadequate interpretation of a falling global ejection fraction or were related to confounding intraoperative myocardial ischemic injury. In any case, the stable ventricular performance coefficients observed in our patients indicate well-preserved global pump function immediately after valve replacement despite partial or complete resection of the mitral apparatus. Late changes in left ventricular configuration or global performance associated with mitral discontinuity (which have been suggested to correlate with a poor long-term survival) were not addressed in this acute study. Follow-up data would need to be acquired more than 6 months after operation after myocardial remodeling has taken place. However, our findings immediately after valve replacement weaken the argument for anterior leaflet-preserving procedures [11, 12, 29, 30], and these results, as well as potential complications related to outflow tract obstruction by the retained anterior leaflet, have prompted us to abandon this procedure completely. Whenever possible, we continue to preserve the posterior leaflet to bolster the prosthetic suture line and potentially reduce the incidence of posterior ventricular rupture. Based on results of this study, however, the conceptual importance of total mitral continuity in preserving left ventricular function during prosthetic valve replacement seems less important. Future investigation will be needed to evaluate the role of the mitral valve apparatus on left ventricular function late after operation.

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DISCUSSION

DR ANDREW S. WECHSLER (Richmond, VA): I compliment Harpole and associates on a very elegant study. Data have been made available to us that were heretofore not obtainable because of the introduction of new technology. Also, I thank Harpole and associates for giving me the paper to review in advance.

My colleagues and I personally are pleased to see these results because they support well some animal experimentation that has been done by David Salter in our laboratory. I anticipate that Dr David is equally displeased to see these results and will probably wish to make some comments.

I have some questions that relate to the data. First, were any of the patients receiving inotropic support or other pharmacological medication at the time of the repeat study?

Second, what are your thoughts as to whether or not the changes you observed at 24 hours compared with the changes you measured intraoperatively had built into them the confounding problem that the patients were not anesthetized and were extubated, thereby making it difficult to compare the postoperative with the intraoperative studies?

Third, what influence might circulating catecholamines have had on the studies done after cardiopulmonary bypass? For example, might they erroneously enhance measurements of contractility regardless of which indices we use?

Fourth, the manuscript indicates that data acquisition occurred at 20-ms intervals. I wonder if that in any way makes it more difficult to determine accurately the end-systolic pressure–volume point, which is used certainly for the calculations of the slope of the end-systolic pressure–volume relationship (E_{max}) and may even play some role in the work load computation, as there is a potential variation of 30 ms on either side of a given arbitrary data point had you, for example, used 5-ms acquisitions as is done with other kinds of technology.

The last question relates to the analysis of the E_{max} data. In thinking about the clinical relevance of these very important observations, it appears that it is easier to test the null hypothesis by using the paired *t* test method. Is it not relevant that in simply looking at your data, there were a number of patients who, in fact, did have a decrease in E_{max}? Even if you are unable to say for the group of patients studied that there is a uniform diminution of E_{max}, would that not match the clinical observation that occasionally after mitral valve replacement, some patients experience unexplained and marked deterioration of left ventricular function?

These are difficult questions, and I realize they exceed the limits of the methods that were used. I again compliment you and your group on a terrific paper.

DR HARPOLE: To answer your first question, I made sure that none of the patients were receiving any inhaled anesthetic agents, and we did not use any kind of inotropic agents during the study period.

Four of the patients did leave the operating room receiving less than 4 μg/kg/min of dopamine, but were weaned in a few hours. No patient was on a regimen of therapeutic iontropic support (5 to 10 μg/kg/min). We attempted to remove the effects of anesthesia and medications from the study.

Skipping to the third question, there is no doubt that cardiopulmonary bypass can itself increase catecholamine levels in patients. That has been shown in multiple studies. We did not measure circulating catecholamine levels in these patients before and after bypass, which certainly would have been helpful. The effect they might have on the results is something I have to accept as a limitation of the study in its existing form. I do not know how we would be able to eliminate that from the protocol using bypass in humans, but you are right: that could affect the data.

Comparing the 24-hour study and the intraoperative study, the 24-hour study was originally placed in the protocol to make sure there was not a drastic change in the load state in these patients when they initially came off bypass and 24 hours after, and so I just included after extubation and after the discontinuation of intravenous medication to see if there was a tremendous change. The fact that we saw no significant change does not eliminate the possibility that there may have been smaller changes. However, we did not see any gross changes in load.

I can say nothing about performance because these patients did not have catheters in the ventricle at 24 hours. Consequently, we were unable to vary preload and get a good preload recruitable stroke work relationship at 24 hours. This is something that we are planning for the future.

The 20-ms acquisition time was the standard time for the RNA volumetric data. We have compared and validated these data against digital subtraction angiographic data that had a 5-ms acquisition time. We had very good results and good comparison for pressure and volume at end-systole. However, there is no question that for the end-systolic pressure–volume relationship, because of the acquisition interval, the upper right-hand corner of the loop could be affected, and that is why I used the preload recruitable stroke work as the standard method of calculating performance. We did calculate the end-systolic/pressure–volume relationship data. However, I agree there could be some variation involved.

Finally, there was some variation in the patient population. Incidentally, the patient whose valve was completely excised was not 1 of the patients whose function decreased postoperatively.

This is a pilot study, it is a small population, and there certainly is some interpatient variability I cannot explain looking at the intraoperative and postoperative courses. With only 12 patients in two groups, I think you are right that it is a type II error. We need a larger population to really come up with more information concerning what is causing the decrease in function postoperatively in these patients.

DR TIRONE E. DAVID (Toronto, Ont, Canada): I find it almost always necessary to excise the entire mitral valve in patients with advanced MS to make it possible to implant a prosthesis of reasonable size. Because we believe that the chordae tendineae are important for ventricular function as well as prevention of spontaneous rupture of the left ventricular wall, we recently began to investigate the effect of preservation of the chordae tendineae in these patients. After excision of the valve we reestablish continuity between the mitral annulus and the papillary muscle heads with synthetic chordae tendineae made of Gore-Tex sutures. Although we have studied only 5 patients as part of a pilot study for a grant proposal, we observed a significant difference in postoperative left ventricular function after 6 months compared with patients who had standard mitral valve replacement.

In patients with mitral insufficiency, I think I agree with your conclusions. Among your 8 patients with mitral insufficiency only 1 had the valve completely excised. One patient had very depressed systolic function. I bet that patient is the 1 in whom you excised the entire mitral valve.

After studying numerous patients during the past decade, we have learned that approximately one third of the patients have normal and two thirds have impaired resting left ventricular function after conventional mitral valve replacement. The reverse occurs when you preserve the chordae tendineae. Thus, although preservation of chordae tendineae is important for left ventricular function, there may be other factors involved.

DR J. D. HARRAH (Huntington, WV): I commend the authors on a fine presentation and have but one question.

I assume these were elective operations. How would you compare the patients who require mitral valve replacement after an acute infarction with your group of patients?

DR HARPOLE: I can only conceptualize or make an educated guess. I think that for patients who have ventricular function compromised secondary to ischemia, most surgeons believe that valve repair is the first procedure of choice if at all possible, that is, the valve has not degenerated to such a point that it cannot be repaired. I agree with that. As to whether or not the patient would show a decrease in function after the procedure, I assume not.

The point we were trying to make with this study is that it seems that much of the change seen when a mitral valve is repaired or replaced for MR is really a change in the load state. We were not able to measure any tremendous global change in performance because the most important change in function is removing the low-impedance atrial outflow tract and forcing all the blood to go through the high-impedance channel through the aorta.

By definition, you are going to have to increase load and because of that, you are going to decrease the global ejection fraction in these patients by forcing all the blood to go the right way.

In these patients with MR, the forward cardiac outputs increased, although the volumetric outputs decreased because the blood was not going back into the left atrium; it was all going out the aortic root.

DR HENRY M. SPOTNITZ (New York, NY): In a group of about 30 patients studied echocardiographically during mitral valve replacement at Columbia-Presbyterian Medical Center, Dr Antunes, Dr Robbins, and I found that the effects of operation on function are dependent on initial ejection fraction. When initial ejection fraction is higher than 40%, ejection fraction drops during operation with little change in contractility. When initial ejection fraction is less than 40%, contractility decreases and ejection fraction does not change much. Left ventricular mass tends to increase more in patients with low ejection fractions, but we also found no significant change overall. In many patients, ejection fraction increases after 24 hours, although not to the preoperative level (unless there is a paravalvular leak). This suggests that part of the observed change in ejection fraction is due to a depression in contractility requiring more than 24 hours to reverse.

We also find a decrease in left ventricular compliance, probably due to the rigid prosthesis in the mitral annulus. What compliance effects did you observe?

Finally, many people who do mitral repair or chordal preservation report that resting ejection fraction does not decrease in the manner you report. What are your comments on this? Does decreased compliance with mitral valve replacement prevent left ventricular shrinkage? Do differences in ejection fraction imply differences in stroke work performance between mitral valve replacement and mital valve repair, even though contractility is unchanged?

DR HARPOLE: First, you are right that in our population, the 24-hour state was not meant to be a late follow-up. That is why I had that disclaimer in the conclusion.

We are, in fact, calling these patients back in. A mean time of about 18 months has elapsed since the procedures and repeat echocardiograms and rest and exercise radionuclide angiocardiograms will be performed in the next 6 weeks. We hope we will be able to see some regression in the mass.

As for the question about compliance, we are just beginning to analyze the data for diastolic function and compliance and hope to have them ready for the American Heart Association meeting.

Finally, I have talked extensively to Dr Rankin and others about the work of Dr David and his colleagues. That group reports that when they have done repairs or replacements, they have seen no change in ejection fraction for the patients having repair or having the mitral apparatus left in place. However, when the valve is excised, they see a decrease. I was having trouble conceptualizing that in considering preload, contractility, heart rate, and afterload as the four things that affect heart function. I think there certainly could be a change in compliance.

The other thing noted in some of the studies is that there was a significant decrease in end-diastolic pressure or end-diastolic volume index when the studies were repeated. Certainly if you decreased preload or decreased afterload—in other words, the mean blood pressure dropped—there would be a consistent ejection fraction, and when and if you reproduced totally the load state before and after, you might see a decrease. I do not have a good explanation for that and would be happy to entertain any ideas from Dr David about that.

Ventricular Septal Defect Associated With Aortic Valve Incompetence: Results of Two Surgical Managements

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Ph. Blondeau, MD, Ch. Dubost, MD, and A. Carpentier, MD, PhD

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Aortic valve insufficiency with ventricular septal defect is usually treated by plication of the commissures. However, long-term deterioration is common. We propose a new technique that corrects the aortic annulus dilatation and the leaflet prolapse and reinforces the sinus of Valsalva. Two groups were compared: group I (29 patients) had plication of the prolapsed leaflet(s) and folding of the free edge; group II (26 patients) had triangular resection of the prolapse cusp, annuloplasty, and reinforcement of the aortic wall. The two groups were similar with regarding to preoperative clinical data. There was no perioperative mortality. Primary failure (aortic valve replacement) occurred in 8 patients in group I (28%) and in 2 patients in group II (8%). The rate of secondary failure was 31% in group I and 4% in group II. The actuarial rate of freedom from reoperation at 5 years is 55% in group I and 88% in group II ($p < 0.05$). The late mortality was 6.5% in group I and 10.9% in group II (no difference). We conclude that aortic valve insufficiency with ventricular septal defect is a malformation of the aortic leaflets, the annulus, and the sinus of Valsalva, and that the proposed technique offers a better result than the usual methods in terms of residual aortic valve insufficiency.

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Ventricular septal defect (VSD) and aortic valve incompetence is a well-known association [1]. Prevention of the aortic valve incompetence is commonly obtained by early closure of a subpulmonary VSD [2–5]. However, the management of serious aortic valve insufficiency is still controversial, especially during childhood and for adults [6, 7].

In 1973, Trusler and associates [8] described a surgical technique that was widely used. The prolapsed cusp was plicated on the commissure areas with intraaortic and extraaortic wall pledgets. Other techniques were described; the basic concept was folding of the free edge of the prolapsed leaflet by a continuous suture [9, 10].

Despite improvements in surgical management, the method of choice remains unclear because of early and late failures [7, 11]. Another approach to aortic valve repair was used in our institution since 1977. Valve repair was achieved by a triangular resection of the prolapsed cusp, reinforcement of the aortic wall, and an aortic annuloplasty as proposed by one of us [12]. The purpose of this study was to compare the results of the later technique, used in 26 patients (group II), with those of the earlier techniques, used in 29 patients (group I).

Material and Methods

Patient Population

From 1963 to 1989, 70 patients were operated on for VSD and aortic valve incompetence at Hôpital Broussais. Six patients had closure of the VSD only. In 9 patients who underwent primary aortic valve replacement, no attempt at valvar reconstruction was made because of active endocarditis (3 patients), calcifications (4 patients), or a bicuspid valve (2 patients).

Fifty-five patients, included in this study, underwent aortic valve repair as a primary procedure. Twenty-three were male and 32 were female. Ages ranged from 2 to 48 years. These patients were separated into two groups according to the surgical techniques.

GROUP I. From 1963 to 1983, 29 patients ranging in age from 4 to 48 years (mean age, 16.5 years) were operated on using current techniques of repair. The preoperative functional status is shown in Table 1.

Aortic valve insufficiency was assessed by aortography and was graded from 1 to 4 [13] (Table 2). There were 4 patients with grade 2, 15 with grade 3, and 5 with grade 4. The angiographic data were not available in 5 cases. Nineteen patients were catheterized before operation. The ratio of systolic pulmonary arterial pressure to systolic aortic pressure ranged between 0.15 and 0.5 (mean, 0.27). The VSD was perimembranous in 11 patients, infundibular in 10, and not clearly defined in 8 (Fig 1).

All patients had a cusp prolapse, involving the noncoronary cusp in 10, the right coronary cusp in 9, and both

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Table 1. Age and Preoperative Condition

Variable	Group I (n = 29)	Group II (n = 26)
Age (yr)*	16.6 ± 12 (4–48)	12.4 ± 6 (2–28)
NYHA		
I	3	1
II	18	17
III	7	6
IV	2	2

* Numbers in parentheses are range.

NYHA = New York Heart Association.

Table 2. Preoperative Evaluation of Aortic Valve Incompetence

Variable	Group I (n = 24)	Group II (n = 26)
Aortic pressure (mm Hg)		
Systolic	117 ± 18	116 ± 14
Diastolic	49 ± 14	52 ± 12
Aortic valve incompetence (grade)[a]		
2	4	2
3	15	19
4	5	5

[a] For group I, grade was determined by angiocardiography; for group II, grade was determined by angiography and Doppler color flow mapping.

cusps in 10 (Fig 2). No correlation was found between the type of VSD and the location of cusp prolapse. Eight patients had associated lesions as follows: right ventricular infundibular stenosis, 4 patients; endocarditis, 2 patients; pulmonary valve agenesis, 1 patient; and mitral valve insufficiency, 1 patient.

GROUP II. From 1977 to 1989, 26 patients, whose ages ranged between 2 and 28 years (mean, 12.4 years), were operated on with the Carpentier technique. The preoperative functional status is shown in Table 1.

Aortic valve insufficiency was assessed by angiocardiography [13] and recently by Doppler color flow mapping [14]. There were 3 patients with grade 2, 19 with grade 3, and 5 with grade 4. The ratio of systolic pulmonary artery pressure to systolic aortic pressure ratio measured in 22 patients was between 0.13 and 0.89 (mean, 0.28). The VSD was infundibular in 13 patients, perimembranous in 10, and not clearly defined in 3 (Fig 1).

All patients had a cusp prolapse, involving the noncoronary cusp in 5, the right coronary cusp in 16, and prolapse of both cusps in 5 (Fig 2). One patient had malformation of the commissure between the right and left cusp. Eleven patients had associated lesions as follows: right ventricular infundibular stenosis, 7 patients; endocarditis of the aortic valve, 1 patient; absence of right superior vena cava, 1; and pulmonary valve agenesis, 1.

Operative Procedure

GROUP I. Operations were performed with numerous types of myocardial protection: moderate hypothermia (28°C) (18 patients), topical cooling (8 patients), selective coronary ostia perfusion (5 patients), and cardioplegia (6 patients). Aortic valve lesions were treated by commissural plication according to the method of Trusler and colleagues [8] in 8 patients, cusp folding in 17 patients [9], and association of both methods in 4 patients. The approach to the VSD was the right ventricle in 5 patients and the aorta in 25. A patch was used in all patients.

GROUP II. Myocardial protection was selective cannulation of the coronary arteries (6 patients), moderate hypothermia (28°C) associated with topical cooling (10 patients), and crystalloid cardioplegia in the coronary sinus (10 patients) [15]. Aortic cusp leaflet prolapse was corrected with a triangular resection [12]. The left coronary cusp was used as a reference to measure the length of the normal free edge. The procedure was repeated for each prolapsed leaflet (Fig 3). The VSD was closed through the aorta with a glutaraldehyde-preserved autologous pericardial patch [16]. At the upper limit of the VSD, stitches were passed in the aortic wall to reinforce the so-called aortic annulus [12] (Fig 4). In subcristal VSD the aortic valve annulus was situated in a lower position than the pulmonary artery annulus and the crista was between these two structures. In infundibular VSD aortic and pulmonary valve annuli were at the same level without any muscular structure between them.

Distention of the annulus was repaired in 20 patients.

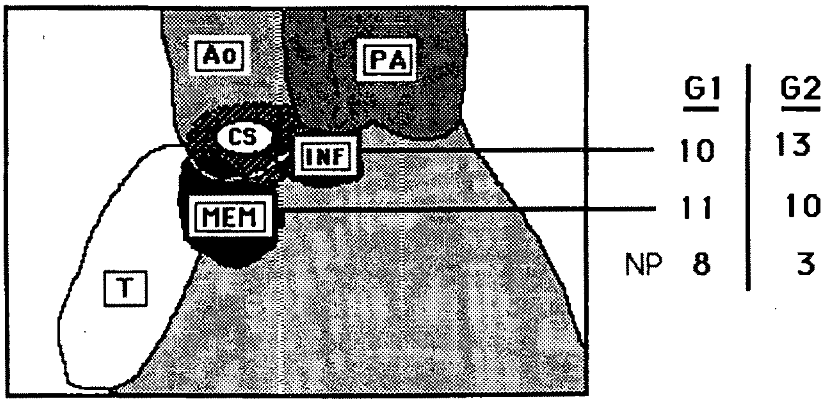


Fig 1. Localization of the ventricular septal defect (VSD) with regard to the conal septum (CS). (Ao = aorta; INF = infundibular VSD; MEM = membranous VSD; NP = VSD not precisely localized; PA = pulmonary artery; T = tricuspid valve.)

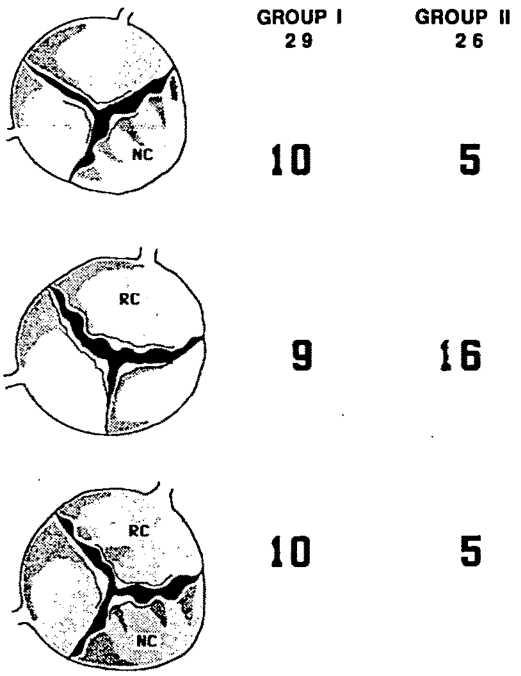


Fig 2. Localization of cusp prolapse. (NC = *noncoronary;* RC = *right coronary.)*

Aortic annuloplasty was performed by a running suture passed up and down the annulus (Fig 5). The circular suture was sized on a No. 23 Hegar dilator for adults. For children, the size of the aortic annulus was based on the tables of Rowlatt and colleagues [17]. The annulus suture material was nonresorbable in adults and resorbable in children [18].

Results

Early Results

There was no operative or perioperative death in either group. One patient in each group had a permanent postoperative third-degree atrioventricular block and a pacemaker was inserted (group I, 3% [70% confidence limits {CL}, 0% to 11%]; group II, 4% [CL, 0% to 12%]) [19].

The aortic valve repair was judged unsatisfactory at operation whenever good coaptation of the leaflet was not obtained or when systemic pressure decreased 20 mm Hg after removal of the aortic clamp. Primary repair failure occurred in 8 patients in group I for an incidence of 28% (CL, 18% to 39%) [16] and in 2 patients in group II for an incidence of 8% (CL, 3% to 17%). A prosthetic valve replacement was used in 3 patients, and a bioprosthesis was used in 5 patients.

Analysis of primary repair failure with regard to the type of repair indicated a significant difference of failure rates in group I and in group II (Table 3). Comparison of primary repair failure in the commissural plication group (13%; CL, 2% to 36%) and in group II (8%; CL, 3% to 17%) showed no significant difference (Table 3).

Late Results

The event-free curve was analyzed by the Kaplan-Meier method [20] and the Log-rank test [21].

GROUP I. Twenty-one patients with valve repair were followed for a postoperative period of 3 months to 21.8 years (mean, 7.6 ± 5.8 years). Nine patients (31%; CL, 21% to 42%) underwent a reoperation for severe aortic valve insufficiency after a delay of between 2 months and 13 years (mean, 6.5 years) (Table 3). Among them, 2 had residual VSD. The causes of aortic valve insufficiency were cusp prolapse (5 patients), cusp retraction (2), calcification (1 patient), and tearing of one leaflet (1).

All patients except 2 underwent aortic valve replacement, with 1 perioperative death and one postoperative atrioventricular block treated by pacemaker implantation. The freedom from reoperation was 60% at 1 year and 55% at 5 years (Fig 6). Two patients who had a second conservative procedure were reoperated on for aortic valve replacement 4 years after the second procedure. Of the 11 surviving patients who had a conservative repair without early or any reoperation, 9 were in New York Heart Association functional class I and 2 were in func-

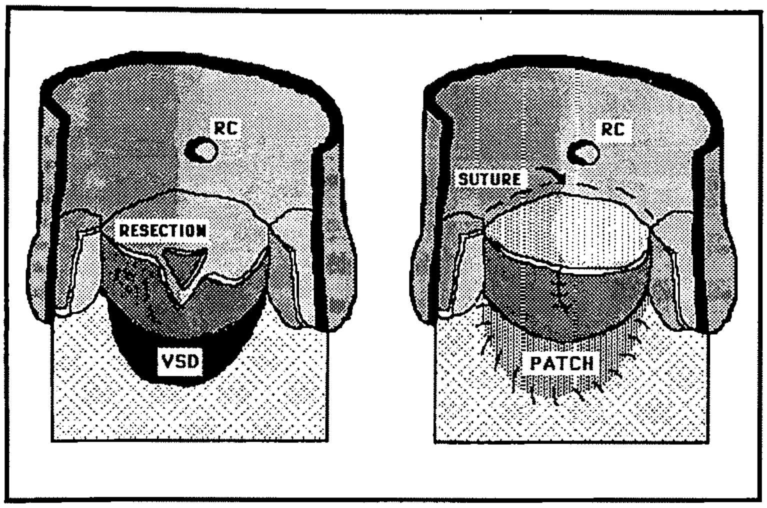
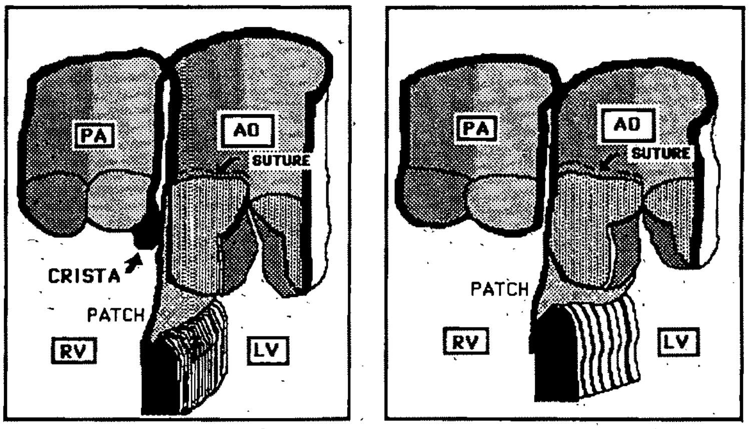


Fig 3. Operative technique in group II: triangular resection of the prolapsed cusp. Closure of the ventricular septal defect (VSD) *with a patch.* (RC = *right coronary artery ostium.)*



SUB CRISTAL VSD **INFUNDIBULAR VSD**

Fig 4. Operative technique in group II: suture of the patch in the aortic wall through the aortic approach to reinforce the sinus of Valsalva. (Ao = *aorta;* LV = *left ventricle;* PA = *pulmonary artery;* RV = *right ventricle.)*

tional class II with no salient complication. Six patients had a grade 1/6 diastolic murmur, 2 had a grade 2/6 murmur, and 1 had a grade 3/6 murmur.

GROUP II. The 24 patients with a valve repair were followed for a period of 6 months to 11.7 years (mean, 2.9 years). One patient was reoperated on for aortic valve incompetence 3 months after the initial procedure and had a valvar replacement.

Two patients died during the follow-up period. The first died 4 years after the operation. The result of the aortic repair was not satisfactory. An associated pulmonary valve agenesis was not treated. A reoperation should have been performed before irreversible intractable congestive heart failure occurred. The second patient died suddenly 4 years after the initial procedure of a cerebral hemorrhage resulting from an aneurysm. He had a normal clinical examination with no heart murmur 1 week before his death. He was maintained by his cardiologist under anticoagulation therapy despite sinus rhythm and satisfactory aortic valve repair.

The freedom from reoperation was 88% at 1 year and remains stable at 3 years (Fig 6).

Twenty-one patients were reviewed for this study. Nineteen were in functional class I and 2 were in class II. Nine patients had no diastolic murmur, 6 had a grade 1/6, 5 had a grade 2/6, and 1 had a grade 3/6 diastolic murmur with no hemodynamic significance (no enlargement of differential pressures).

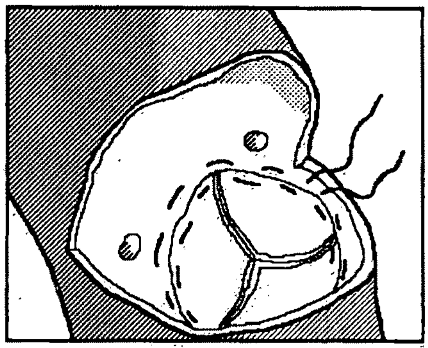


Fig 5. Circular annuloplasty.

Comment

Different classifications have been proposed for aortic valve insufficiency with VSD according to the location of the VSD [1, 22] (subcristal or subpulmonary), the presence of an infundibular stenosis [1], and the presence of a conal support to the aortic annulus [23]. All these classifications were based on anatomical studies, taking into consideration the aspect of the VSD as viewed from the right ventricle. From this study, Carpentier proposed a simpler and more convenient approach, taking into account only the aortic view of the VSD because this lesion is now treated through the aortic root. Important consideration was given to the fact that not only the cusps and the ventricular septum but also the sinus of Valsalva was malformed. In most cases, the sinus of Valsalva has two structural parts. The upper part is made of aortic wall tissue, the lower part of leaflet tissue. Those two parts are in continuity and are not separated by a true fibrous annulus. As a result of this configuration, the cusp itself

Table 3. Incidence of Primary and Secondary Failures

		Primary Failure			Secondary Failure		
Group	No. of Patients	No.	%	70% CL (%)	No.	%	70% CL (%)
I	29	8	28	8–39	9	31	21–42
Commissural plication	8	1	13	2–36	2	25	9–50
Cusp folding	17	7	41	27–57	5	29	17–45
Commissural plication and cusp folding	4	0	0	0–38	2	50	18–82
II	26	2	8	3–17	1	4	0–12

CL = confidence limits.

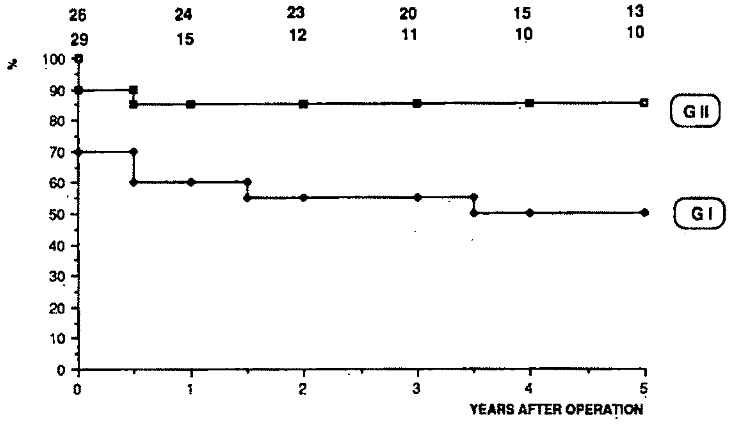


Fig 6. Freedom from valve replacement or reoperation (analyzed by Kaplan-Meier and Log-rank test).

is much longer than normal and has two "functional" parts: one "parietal," which tends to correct the VSD during diastole, and the other "luminal," which functions as a cusp. The valvar insufficiency results from the primary billowing of the cusp through the VSD during diastole and to secondary lesion, namely cusp prolapse resulting from a progressive distention of the free edge of the cusp. The commissures are usually normal, and the predominant lesions are located on the midportion of the cusp.

The aim of the reconstructive operation is to close the VSD and reconstruct the sinus of Valsalva to support the prolapsed cusp and to correct the cusp prolapse by a triangular resection. The final step is to correct the annulus aortic dilatation, which can be an important factor in the aortic incompetence. This approach differs from the classic techniques in that the surgical procedure involves the areas of maximum lesions, ie, the medial part of the cusp rather than the commissures, which are usually normal and therefore must be preserved. It also avoids some of the drawbacks that attend the current techniques, ie, cusp deformation, fibrotic retraction, cusp tears at commissural stitches, and recurrent annulus distention [7, 11].

Comparison between the two groups indicates a lower rate of early and late valve deterioration in group II. Clinical characteristics of the patients were similar in both groups. A temporal bias in the comparison is possible because the dates of operation were different in the two groups; however, evaluation of the aortic valve repair was performed with the same criteria to obtain a good result.

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DISCUSSION

DR GEORGE A. TRUSLER (Toronto, Ont, Canada): I congratulate Chauvaud and associates on an excellent presentation. I was privileged to read the paper and it will certainly add to our understanding of aortic valve repair.

I was perplexed, however, by the poor results of the first group. In our series of 70 patients, freedom from reoperation was 89% at 5 years and 86% at 10 years. There were several differences in the patients in the present series, including an older mean age and a more frequent involvement of the noncoronary cusp, both of which would have an effect.

Perhaps the technique was also different. Pure aortic valve prolapse can be eliminated if the elongated cusp margin is accurately measured and securely plicated. The pledget inside the aorta overlaps the upper border of the plication. The tiny Dacron hood anchors the repaired cusp to its neighbor. These details may be important only occasionally but are sometimes overlooked.

The "Carpentier repair" produced good results with a freedom from reoperation of 88% at 5 years. A triangular resection in the middle of the cusp seems courageous, but obviously works. In our experience, the thin weak portion of cusp is usually near the commissure, and we try to eliminate this in the plication. Certainly, in the infrequent presence of serious central fibrosis and retraction, a triangular resection is indicated.

We agree that reinforcement of the aortic sinus is sometimes necessary, particularly in older patients and, when patching the VSD, we anchor the upper border securely, above any prolapse.

Similarly, I suspect that Chauvaud and associates are right in saying that some patients have aortic root dilatation, but it is not clear how many of these patients require annuloplasty; we have not performed it and indeed did not find older age to be a risk factor for valve failure.

Dr Chauvaud, in retrospect, can you account for the results in your first group? Have you any concrete evidence that the root annuloplasty was essential to success?

DR CHAUVAUD: I see two reasons for the poor results of group I. First group I was the earliest in the Broussais Hospital experience, and the operative failures may have been the result of the learning curve in this disease. Second, in group I, surgical techniques were mixed and included commissural plication and folding of the free edge. Because of the midterm deterioration of the folding technique, we gave up this method.

With regard to the question about aortic root annuloplasty, I think that annuloplasty is mandatory in "old" patients. In our country, we still operate on children and adults with massive aortic annulus dilatation. Thus, in some patients we try to avoid annuloplasty and later find we must perform aortic annulus reduction because of residual aortic valve incompetence. We have used aortic annuloplasty in 26 patients.

Clinical Experience With Minitracheostomy

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Minitracheostomy is a technique to assist in the removal of airway secretions while maintaining glottic function. A flanged, reclosable cannula 4.0 mm in internal diameter is inserted through the cricothyroid membrane into the trachea. Sixty procedures were performed in 56 patients from July 1988 to June 1989. Indications for placement included excessive postoperative secretions (46/60), difficulty with endotracheal suctioning (4/60), preoperative secretions (4/60), postpneumonic secretions (5/60), and acute airway obstruction (1/60). Successful intratracheal placement was possible in all instances, and the device was well tolerated. Major intratracheal bleeding necessitating endotracheal intubation occurred in 2 patients. Minor complications included local hematoma (5 patients), subcutaneous emphysema (2 patients), and hoarseness (1 patient). No deaths occurred. Cannulas remained in place for one day to 35 days. Removal resulted in closure within 48 hours. No adverse laryngeal effects were seen. A successful result, not requiring other invasive methods to remove secretions, was obtained in 43 (72%) of the 60 procedures. Minitracheostomy is a useful adjunct for secretion removal in the hospitalized patient.

(Ann Thorac Surg 1990;49:881–6)

Maintenance of bronchopulmonary hygiene is the primary goal of pulmonary therapy for the hospitalized patient. Removal of secretions from the tracheobronchial tree is crucial to achieving this goal. Therapies designed to assist in the removal of airway secretions include chest physical therapy, incentive spirometry, endotracheal suctioning, and bronchoscopy.

Minitracheostomy was first described by Matthews and Hopkinson [1] as a novel method for endotracheal suctioning with preservation of glottic function for cough and phonation. The technique involves placement of an indwelling plastic cannula 4.0 mm in internal diameter into the trachea through the cricothyroid membrane. The cannula allows reliable, convenient aspiration of secretions from the large airways. Because glottic function is maintained, use of the minitracheostomy also effectively stimulates a cough. The minitracheostomy cannula can also be used for intratracheal administration of medications, for airway access in acute obstruction [2], or for access for jet ventilation [3].

This report describes our initial clinical experience with minitracheostomy from July 1, 1988, to June 1, 1989, in an attempt to assess the effectiveness and safety of this technique.

Material and Methods

Any patient with copious tracheobronchial secretions, ineffective cough, or both and any patient requiring frequent chest physical therapy, "blind" endotracheal suctioning, or flexible bronchoscopy was considered a candidate for minitracheostomy. Patients with ongoing aspiration of oropharyngeal secretions or patients without an intact gag reflex were excluded. Patients with an infiltrate on chest roentgenograms that was thought to represent lobar pneumonia were also excluded. Systemic anticoagulation was considered an absolute contraindication to placement. A qualitative coagulopathy (eg, as in renal failure) or the use of subcutaneous heparin sodium or salicylates was not a contraindication to minitracheostomy.

All patients identified as candidates for minitracheostomy were included in the study. The indication for placement as well as complications with the procedure, duration of cannula placement, and patient outcome were recorded. Frequency of cannula use by the nursing staff was noted, as was the subjective response of the patients to the device. Fiberoptic bronchoscopy and methylene blue tests (to assess aspiration) were performed as clinically indicated.

Anatomical landmarks, including the thyroid notch, cricoid cartilage, and cricothyroid membrane, were identified with the neck in extension. After sterile preparation of the anterior cervical region, local anesthetic (typically, 3 to 5 mL of 2% lidocaine hydrochloride with epinephrine) was infiltrated over the proposed site of incision in the midline of the cricothyroid membrane (Fig 1). Placement of the cannula was through a vertical incision 3 to 5 mm in length over an obturator, as described by Matthews and Hopkinson [1]. A commercially available kit (Mini-Trach II; Portex, Concord, NH) containing a reclosable, flanged cannula, obturator, beveled knife blade, 15-mm adaptor, 10F suction catheter, and tracheostomy tape was used. Although a Seldinger method for placement of the cannula has also been described [4], this approach was not used.

Intratracheal placement was confirmed by listening for air exchange through the proximal end of the cannula, by aspiration of tracheobronchial secretions, by initiation of cough by stimulation of the carina, or by flexible bron-

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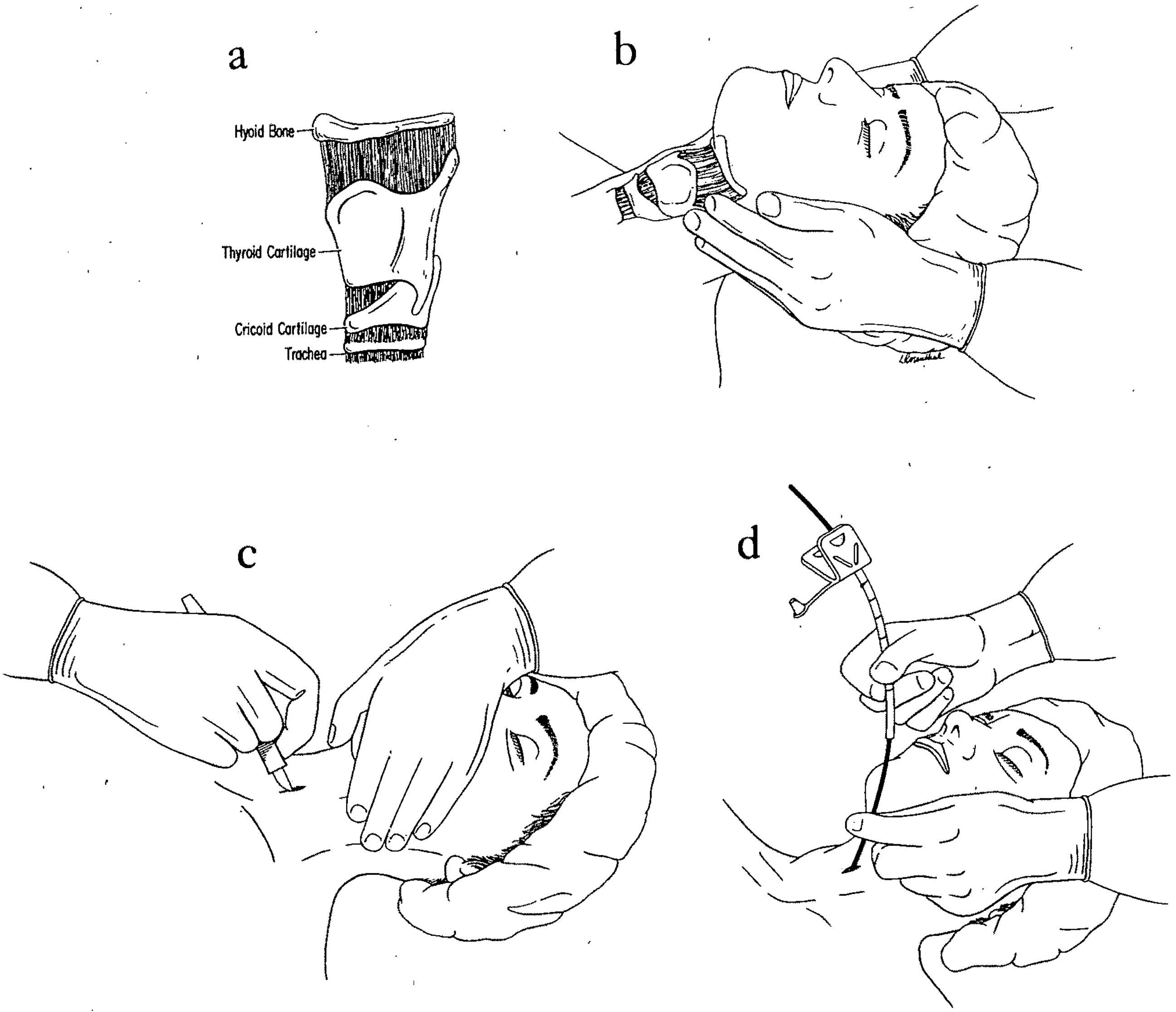

Fig 1. Minitracheostomy insertion technique. (a) Anatomy of the upper airway. The cannula (outer diameter, 5.4 mm) is inserted through a vertical incision in the cricothyroid membrane. (b) The head is positioned with the neck in extension, and the thyroid notch, cricoid cartilage, and cricothyroid membrane are identified. (c) After instillation of local anesthetic, a vertical incision in the cricothyroid membrane is made with the bevel of the knife directed caudally. Egress of air should be identified with successful incision into the subglottic space. (d) The obturator is passed through the incision; its curvature directs it distally into the trachea. Resistance should be minimal. The cannula is guided over it into the trachea. After removal of the obturator, intratracheal placement of the minitracheostomy is confirmed by respiration through the cannula, aspiration of airway secretions, and elicitation of cough by direct stimulation of the carina.

choscopy. The cannula was secured with a standard tracheostomy tape after placement.

Aspiration of secretions through the cannula was done using 10F suction catheters. A small amount of saline solution (3 to 5 mL) was instilled before each use of the minitracheostomy to stimulate cough and improve secretion mobilization. In the first 24 hours after placement, the cannula was typically used to aspirate secretions every two hours. For the next 24 hours, the cannula was used every four hours. Subsequently, the device was used as determined by clinical assessment of the patient. When not in use, the cannula was kept closed with the attached cap to prevent loss of humidification and inspissation of secretions.

Results

A total of 56 patients were evaluated for minitracheostomy placement. Patients were obtained from the general thoracic (24/56, 43%), cardiovascular (9/56, 16%), and general surgical (18/56, 32%) services or from the general medical services (5/56, 9%). Prior surgical procedures included thoracotomy (20 patients), esophagectomy (8), coronary artery bypass grafting (8), thoracic or abdominal aortic aneurysm repair (7), gastrointestinal procedures (7), and drainage of a subdural hematoma (1 patient).

A total of 60 minitracheostomy procedures were performed. Two patients required placement of a second cannula, and 1 patient underwent three successive mini-

Table 1. Indications for Placement

Indication	No. of Placements
Retained secretions	
Postoperative	46
Preoperative	4
Postpneumonic	5
Difficulty suctioning	4
Acute airway obstruction	1
Total	60

tracheostomies. In patients who had multiple procedures, the minitracheostomies were performed at discrete intervals for different indications during the hospital course, and not for malfunction of the device.

The main indication for minitracheostomy placement was excessive postoperative secretions (46/60, 77%) (Table 1). In 25 patients extubated immediately after operation, the minitracheostomy was placed a mean of four days postoperatively (range, one to nine days). In 21 patients requiring ventilation for a period after operation, minitracheostomy was performed a mean of two days after extubation (range, immediately after extubation to five days after extubation).

Other indications for placement included excessive postpneumonic secretions (5/60, 8%), excessive preoperative secretions (4/60, 7%), and difficulties with standard blind endotracheal suctioning (4/60, 7%) related to patient discomfort or anatomical constraints (eg, deviated nasal septum) (see Table 1). One patient with acute airway obstruction underwent minitracheostomy intraoperatively as a temporizing measure until formal tracheostomy could be performed.

Successful intratracheal placement of the cannula was possible in all instances. Availability of equipment for emergency intubation was the only requirement of the facility where insertion was performed. The procedure was carried out at the bedside in the intensive care unit in 32 instances and on the medical-surgical ward in 28. The minitracheostomy procedures were performed by members of the attending surgical staff (23/60, 38%), cardiothoracic fellows (29/60, 48%), and general surgical residents (8/60, 13%).

Complications occurred with ten of the 60 insertions. There was one major complication, which occurred twice: substantial intratracheal bleeding necessitating emergency intubation. The remaining complications were minor and self-limited, and did not interfere with use of the device. Minor bleeding at the incision or localized hematoma was seen in 5 patients, self-limited cervical subcutaneous emphysema in 2 patients, and hoarseness in 1 patient. No complications related to extratracheal cannula placement were noted. No deaths were due to the procedure or any of the observed complications.

Nursing personnel in all areas of the hospital were able to successfully use the cannula for suctioning. No difficulties with care and maintenance of the device were encountered despite wide variations in nursing background and experience.

The cannula was well tolerated by the patients. It caused no interference with phonation, breathing pattern, or swallowing function in any patient. All patients described minor discomfort at the insertion site during the first 24 hours after placement. Subsequently, no discomfort was noted with quiet breathing after 57 of the 60 procedures. Of the 3 patients who complained of discomfort with quiet breathing, 2 tolerated this while the cannula was in place. The other patient complained, and the persistent complaints eventually led to removal of the device, although the cannula was successfully used for 13 days.

Mild discomfort or a choking sensation was noted by all patients with motion of the cannula flange during suctioning or routine nursing care of the device. All patients, however, described less discomfort with use of the minitracheostomy for suctioning compared with blind endotracheal suctioning. All patients continued routine oral intake after insertion. Three patients underwent methylene blue testing while the cannula was in place, and no aspiration was identified.

The minitracheostomy cannulas remained in place for one day to 35 days. The mean duration of placement was eight days and the median duration, six days. No wound complications were noted, and all sites closed within 48 hours of removal of the device.

The minitracheostomy was deemed successful if bronchopulmonary hygiene improved sufficiently to allow eventual removal of the cannula without institution of other, more invasive measures for secretion removal. By this criterion, the minitracheostomy was successful in 43 (72%) of the 60 procedures. Unsuccessful outcomes included those patients requiring endotracheal intubation (12/60, 20%) or formal tracheostomy (5/60, 8%) because of progressive respiratory failure or aspiration. Patients requiring formal tracheostomy after prior minitracheostomy posed no additional technical difficulty related to prior use of the cannula.

Comment

Clearing secretions from the airway is crucial in maintaining bronchopulmonary hygiene. Despite improvements in patient selection, in anesthesia and operative techniques, and in the overall morbidity and mortality rates for surgical patients, pulmonary complications remain the most frequent cause of postoperative morbidity and mortality. Pulmonary complications occur in as many as 75% of patients [5] and account for as much as 75% of the reported mortality for surgical procedures [6, 7]. Inadequate removal of pulmonary secretions predisposes to atelectasis, invasive pulmonary infection, and respiratory failure [8]. Multiple factors in the hospitalized patient predispose to inadequate clearance of secretions. For surgical patients, postoperative pain and mechanical impairment of pulmonary function and expectoration pose additional hazards [5, 9]. Methods to assist with removal of airway secretions are helpful in limiting the impairment of bronchopulmonary hygiene and the attendant morbidity and mortality in these patients.

Standard methods for removal of secretions from the airways include chest physical therapy, incentive spirometry, blind endotracheal suctioning, and bronchoscopy. More invasive procedures, such as endotracheal intubation or tracheostomy, have also been recommended to prevent progressive pulmonary insufficiency [10]. These techniques involve progressive degrees of surgical invasion, with greater patient discomfort and potential for complication. In addition, the more invasive procedures, by eliminating glottic function, deprive the patient of intact, active physiological mechanisms (eg, cough) that assist in clearing secretions and preserving lung function. Minitracheostomy, although more invasive than blind suctioning or perhaps bronchoscopy, preserves glottic function and provides a more reliable and convenient access to the airway [1].

In this series, minitracheostomy was performed for patients with copious tracheobronchial secretions. Typically, the cannula was placed within three days of either operation or extubation and remained in place for a mean of eight days. Placement was successfully achieved in all instances by surgical personnel of varying experience. Patients recovering from an operation were the largest group to have the procedure. This select group of patients, requiring frequent assistance to clear airway secretions, accounted for fewer than 10% of the patients undergoing major surgical procedures. Yet these patients were typically those with limited pulmonary reserve and at highest risk for sputum retention and progressive respiratory failure. For such patients, minitracheostomy was a useful technique to remove secretions and to stimulate the cough reflex.

Of the 60 minitracheostomies performed, 72% had a successful outcome as judged by a continual improvement in bronchopulmonary hygiene. No additional invasive measures were needed for these patients, and the cannula was eventually removed without incident. Minitracheostomy placement was successful in patients recovering from an operation, in patients in whom standard blind suctioning could not be performed, and in patients with excessive postpneumonic secretions. However, placement of the cannula in an attempt to improve secretion status preoperatively was successful only 50% of the time (two of four instances). One patient with amyotrophic lateral sclerosis and 1 patient with a large chronic bronchopleural fistula required endotracheal intubation before operation. Accurate identification of anatomical landmarks is essential before a minitracheostomy is performed.

Major complications were rare, occurring in two of 60 procedures. Both were related to intratracheal bleeding, presumably due to injury of the longitudinal veins associated with the thyroid isthmus, the anterior jugular veins, or both. In these two instances, immediate removal of the cannula when excessive bleeding at the incision was noted resulted in hemorrhage into the airway necessitating emergent reintubation. Provided the cannula is intratracheal, the minitracheostomy is now left in place when bleeding from the incision is identified. Conservative measures, including elevation of the head of the bed, direct pressure, or local infiltration of dilute vasoconstrictive solutions, have controlled all subsequent instances of hemorrhage associated with minitracheostomy. Successful management of bleeding with conservative measures has also been noted by previous authors [1, 11]. Others [12, 13] have reported both extratracheal placement and endotracheal migration of the cannula. Careful attention to anatomical landmarks during placement, as well as use of the flanged cannula, should prevent such occurrences.

Bronchoscopic evaluation while the cannula was in place showed minimal mucosal fibrosis at the puncture site. No long-term airway complications have been observed in follow-up of a minimum of 4 weeks. These results concur with the prospective findings of Campbell and associates [11].

A minitracheostomy is *not* equivalent to a cricothyroidostomy. The relative size of the minitracheostomy cannula (inner diameter, 4.0 mm; outer diameter, 5.4 mm) is much less than the average width of the cricothyroid membrane in adults (8.5 mm). Therefore, the minitracheostomy cannula does *not* provide a sufficient airway for spontaneous, nonassisted ventilation in cases of complete glottic obstruction. Although minitracheostomy was used to gain urgent control of an obstructed airway in 1 patient in this series for delivery of oxygen, limited ventilation was possible only with positive-pressure assistance while formal tracheostomy was being urgently performed.

Minitracheostomy is a safe and effective method for the removal of secretions from the airway. It allows maintenance of glottic function and is well tolerated by patients. In hospitalized patients, particularly patients who are recovering from an operation, minitracheostomy is an invaluable adjunct to standard methods of improving bronchopulmonary hygiene.

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DISCUSSION

DR THOMAS R. J. TODD (Toronto, Ont, Canada): It is always a pleasure to listen to a paper from Massachusetts General Hospital, and it is a particular honor to initiate the discussion.

The concept of minitracheostomy is not new, and there are at least seven reports in the literature detailing its use. Despite the apparent advantages, it has yet to gain popular acceptance in our institutions. I think there are three reasons for this, and these three reasons will generate three questions for Dr Wain.

First, our patterns of practice, our complements of patients, and the availability of auxiliary personnel such as respiratory therapists and physical therapists vary from institution to institution. Thus anecdotal reports are not strictly comparable, and none of the reports to date present either a control trial or definable entry criteria.

The entry criterion in the study of Dr Wain and associates is copious tracheobronchial secretions, and depending on your subjective level of evaluation that day and your clinical experience, that could range from 10% to 50% of patients recovering from an operation.

In our institution, we use fiberoptic bronchoscopy whenever there is major atelectasis secondary to retained secretions. We usually have to do it only once, and it seems to achieve the same objective.

In the manuscript, which I was privileged to read ahead of time, Wain and associates indicate that bronchoscopy was undertaken when clinically indicated, but there are no details as to how often it was done or what constituted a clinical situation. So my first question is, how often did you undertake bronchoscopy and for what reasons?

The second reason for nonacceptance of this technique lies in the fact that there is an absence in the literature of definable end points of success. This is the first paper of which I am aware in which the end point is accurately defined; it is defined as the ability to remove the minitracheostomy without the need for subsequent oral endotracheal intubation. With that criterion of success, Wain and associates quotes a success rate of 72%, or conversely, 28%, or 17 procedures, did require subsequent mechanical ventilatory assistance.

My only problem with that is that there were 21 patients in the group who had ventilation for a time before the minitracheostomy was undertaken. So, my second question is, how many of your 17 failures came from the group who required ventilation for a prolonged period before the tracheostomy was performed? If that number is large, then we could conclude that the requirement of minitracheostomy really reflected extubation that was undertaken too early.

My third problem with the literature lies in the poor definition of respiratory function at the time the minitracheostomy was undertaken, and this seems to be ubiquitous. My third question is, can you give us any data concerning the respiratory function at the time you did the tracheostomy? Did you measure arterial blood gases, vital capacity, or negative inspiratory force before the minitracheostomy was performed?

In summary, the presentation of Wain and associates addresses several of the concerns that I have expressed. It is the only paper of which I am aware that provides long-term follow-up to assure us that there are no airway sequelae from this bedside procedure. The Mass General data are important in that they agree with the consensus in the literature, but most important of all, in my opinion, the data come from individuals whose clinical judgment in this area is impeccable. That lends great weight to any anecdotal report.

DR JACOB B. SHAMMASH (Springfield, MA): I enjoyed this paper; it adds to our armamentarium of aggressive pulmonary aids. Considering the sick patient in the intensive care unit, however, we cannot help but realize that we have added one more invasive procedure, one more line, if you will, to the long list of invasive ports, and one more source of sepsis. Handled carefully, however, this should not be a problem. The procedure cleanses the trachea and possibly the major bronchi. Most patients with retained secretions need a thorough cleansing of the distal tracheobronchial tree and the segmental and subsegmental basilar bronchi.

My colleagues and I have adopted a program of routine bronchoscopy of patients immediately after a major thoracotomy, especially patients with chronic obstructive pulmonary disease who are undergoing pulmonary resection. The procedure is done in the operating room before extubation, and adds only a few minutes to operating time. We call it "quickie cleansing" bronchoscopy. It gives patients a good start on bronchial secretions in the immediate recuperative period. We also use cleansing bronchoscopy at the bedside without anesthesia whenever it is needed in the postoperative period. It has served us well.

DR JOHN R. BENFIELD (Sacramento, CA): Quite properly, this important paper by Wain and associates stressed the role of prevention. I am a little surprised that they did not indicate something about the use of epidural analgesia, which for us has made a tremendous difference in the incidence of postoperative pulmonary complications.

Dr Wain, what was the incidence of epidural analgesia among your patients who required minitracheostomy?

DR HOWARD S. BROWN (Atlanta, GA): Dr Wain, do you have any experience with using prolonged ventilation, say a jet ventilator, as opposed to doing a conventional tracheostomy or a prolonged endotracheal intubation?

DR WAIN: Dr Todd, we typically perform flexible bronchoscopy at least once and usually twice before placing the minitracheostomy. Not uncommonly, perhaps 70% to 80% of our patients who are recovering from a thoracic procedure undergo at least one flexible bronchoscopy during the postoperative period, not on a routine basis but for the indications that you outlined.

As for the patients in whom minitracheostomy failed after extubation after surgical procedures, I do not have the precise numbers on that. In general, though, the failures did not appear to segregate to one group or the other, ie, the group in whom the minitracheostomy was placed immediately after an operation and the group who required postoperative ventilation for a time.

Finally, as to objective data before minitracheostomy placement, we, too, often questioned what would be the most objective data we could obtain. Basically, we just threw up our hands and left it to clinical judgment. So we do not have those objective data you requested, but I certainly think collecting them would be very useful.

Dr Shammash, as I mentioned, we perform bronchoscopy in a large percentage of our patients only for clinical indications. As far as I am aware, routine bronchoscopy has not been shown objectively to cause any improvement in postoperative pulmonary function.

In response to Dr Benfield's question about epidural analgesia, over the time of this study, we used epidural analgesics to a greater extent. In our hospital, however, their use is confined to intensive care units. Most of our patients typically are discharged from the intensive care unit the day after operation, and the epidural agent is removed. In the next year or two, we will probably have epidural analgesics on the floor, and we can address the question about their use in preventing postoperative pulmonary difficulties.

Finally, in answer to Dr Brown's question about jet ventilation, we have not used this device for jet ventilation access. However, Dr Matthews and his group have developed a ventilator that provides proper humidification and that they use quite successfully for jet ventilation through the minitracheostomy. Dr Todd pointed out just before this presentation that there are also jet ventilators that have just come on the market in the United States that can be used for that purpose.

Shone's Anomaly: Operative Results and Late Outcome

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Shone's anomaly, a congenital cardiac malformation, consists of multiple levels of left heart obstruction including supravalvar mitral ring, parachute mitral valve, subaortic stenosis, and coarctation. The prognosis for patients with Shone's anomaly is poor. To assess operative results and late outcome, we reviewed the records of 30 consecutive patients seen with Shone's anomaly at our institution between 1966 and 1989. Anatomical diagnoses in these patients were supravalvar mitral ring (22 patients), mitral valve abnormalities including parachute mitral valve, fused chordae, or single papillary muscle (26 patients), subaortic gradients (26 patients), and coarctation (29 patients). Nineteen patients had all four lesions. Other common defects were bicuspid aortic valve (19 patients) and ventricular septal defect (20). Two patients were treated medically. The other 28 patients required 84 operative procedures with 18 patients undergoing more than one procedure. Operations included coarctation repair (28 patients), mitral valve repair or replacement (11), ventricular septal defect closure (8), subaortic resection (8), and complex left ventricular outflow tract reconstruction or bypass (4). Age at first operation ranged from 7 days to 7 years (median age, 3 months). There were no operative deaths at the first operation. However, mortality rose to 24% (4/17) after the second operation. All operative deaths were secondary to severe mitral valve disease. The survivors have been followed from 1 to 16 years (mean follow-up, 6 ± 1 years). There were no late or sudden deaths. Morbidity has included stroke (1), gastrointestinal bleeding (2), permanent heart block (1), and persistent congestive heart failure (6). We conclude that although patients with Shone's anomaly are seen with a wide spectrum of major anatomical and hemodynamic abnormalities, a satisfactory long-term outcome is possible for most. Operative mortality is adversely affected by the severity of mitral valve disease and the need for multiple operative procedures.

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Since the first description of the developmental complex of parachute mitral valve, supravalvar ring of the left atrium, subaortic stenosis, and coarctation of the aorta by Shone and associates [1] in 1963, little clinical experience has accumulated about the operative therapy or late follow-up of patients with Shone's anomaly. Cardiologists and cardiac surgeons have reported patients with Shone's complex to have an unsatisfactory operative outcome and a poor long-term prognosis.

Although patients with Shone's anomaly tend to be grouped together in terms of therapeutic approach, these patients actually demonstrate a wide spectrum of developmental malformations. We postulated that if the operative approach to these patients is tailored and individualized, the long-term outcome might be better. To assess the operative results and the late outcome with such an approach, we retrospectively reviewed the data from 30 consecutive patients with the diagnosis of the developmental complex of Shone's anomaly.

Material and Methods

The records of 30 consecutive patients seen at The University of Michigan between 1966 and 1989 were examined for the severity of Shone's anomaly, the operative approach, and the eventual late outcome. Reviewed at this time were operative notes, cardiac catheterization reports, inpatient notes, and referring letters. The patients were followed up by a combination of review of outpatient clinic notes and consultation with each patient's private physician.

There were 16 female and 14 male patients. Height and weight at the first operation were 74 ± 9 cm and 8.0 ± 1.7 kg, respectively. Most patients fell between the fifth and tenth percentile for height and weight at the time of first presentation.

Seventeen of the patients were first seen at The University of Michigan in frank congestive heart failure (CHF) necessitating hospitalization. Sixteen of the 17 were on a regimen of digitalis and a diuretic at the time of admission. The condition of 2 additional patients was well controlled with digitalis and a diuretic without evidence of CHF at the time of first admission. Five patients had cardiomegaly on the chest roentgenogram without physical signs of CHF. Four patients had an unremarkable chest roentgenogram and only a murmur on physical

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examination, and 1 patient with a normal chest roentgenogram was cyanotic on admission.

Twelve of the 18 patients who underwent a second operative repair were in CHF. Nine of these 12 patients were receiving digoxin, a diuretic, a vasodilator, or a combination of these before admission.

All patients had systolic ejection murmurs greater than 3/6. Four associated syndromes were detected in 3 patients: adrenogenital syndrome, Turner's syndrome, phocomelia, and "floppy baby syndrome."

Normal sinus rhythm was demonstrated in 28 patients, and the mean axis was 93 ± 8 degrees. One patient was noted to have first-degree heart block. Seven patients had electrocardiographic evidence of left ventricular hypertrophy, 7 patients showed right ventricular hypertrophy, and 9 patients demonstrated biventricular hypertrophy.

Catheterization Data

Right and left heart catheterization data obtained in 26 children were compared. Two patients could not undergo left heart catheterization because of technical difficulties, and the records of 2 patients were incomplete.

The pressures (mean ± standard error of the mean, systolic/diastolic) for each cardiac chamber were as follows: right atrium, 5 ± 1 mm Hg; right ventricle, 62 ± 7/7 ± 1 mm Hg; pulmonary artery, 60 ± 5/24 ± 3 mm Hg; left ventricle, 135 ± 8/13 ± 2 mm Hg; ascending aorta, 114 ± 7/67 ± 4 mm Hg; and descending aorta, 86 ± 5/60 ± 4 mm Hg. Systolic gradients at the area of subaortic obstruction ranged from 10 to 100 mm Hg (mean gradient, 21 mm Hg) and coarctation gradients ranged from 12 to 75 mm Hg (mean gradient, 28 mm Hg). The oxygen saturation was as follows: right atrium, 72% ± 1%; right ventricle, 75% ± 1%; pulmonary artery, 76% ± 2%; left ventricle, 94% ± 1%; and aorta, 89% ± 3%.

Anatomy

Anatomical evaluation was done in all patients by echocardiography, cardiac catheterization, or both, and then confirmed at operation. The aspects of Shone's anatomy for each patient are shown in Table 1. A diagrammatic representation of the four obstructive lesions in Shone's anomaly is shown in Figure 1. Coarctation of the aorta, found in 29% of the 30 patients, was the most common defect. Subaortic stenosis due to a fibromuscular hypertrophy (25 patients) or a subvalvar membrane (1 patient) was also observed. Abnormalities of the mitral valve were seen in 26 patients and included mitral stenosis, fused chordae, single papillary muscle, and parachute mitral valve apparatus. A supravalvar mitral ring was identified in 22 patients.

Nineteen patients had all four components of Shone's anomaly. These were the patients with so-called pure Shone's anomaly. Other associated defects included ventricular septal defect (VSD) (20 patients), patent ductus arteriosus (12), and bicuspid aortic valve (19).

There were 2 patients with additional left-sided malformations noted at catheterization. One patient had a hypoplastic left ventricle and the other, a severely hypoplastic aortic arch. Other valvar abnormalities included moderate tricuspid regurgitation (3 patients) and mild pulmonary stenosis (2).

Operative Data

Of the 30 patients with Shone's anomaly, 28 underwent operative intervention. The operative procedures are summarized in Table 2. Two patients were treated medically. One patient, an 8-year-old child, was considered to have only minimal hemodynamic abnormalities, despite the presence of all four lesions of Shone's anomaly. The other patient, a 3-month-old infant, was considered to have defects that could not be corrected by operation.

Age at the first operation ranged between 7 days and 7 years (mean age, 11 months; median age, 3 months). Nineteen patients had the first operation before 1 year of age. There were 42 procedures performed at the first operation in 28 patients, an average of 1.5 ± 0.2 per patient. The most common procedure was coarctation repair (23 patients), followed by ligation of a patent ductus arteriosus (10). Other procedures at first operation included mitral valvotomy (3), aortic valvotomy (2), and atrial septostomy, pulmonary artery banding, subaortic resection, and a Blalock-Taussig shunt (1 each). Aortic cross-clamp time was 27 ± 4 minutes for procedures requiring cardiopulmonary bypass.

The second operative procedure, necessary in 18 patients, was performed at a mean age of 46 months (range, 1 month to 8 years). The average number of procedures was 1.6 ± 0.4 (28 procedures in 17 patients). These operations included VSD repair (7 patients), reoperation for coarctation (4), aortic valvotomy (3), mitral valvotomy (2), subaortic resection of a muscular band (n = 2), and left ventricular outflow tract myotomy, excision of a subaortic membrane, placement of a Hancock bioprosthesis in the mitral position, descending aorta–main pulmonary artery shunt, apicoaortic conduit, pulmonary artery banding, Konno aortoventriculoplasty with St. Jude aortic valve replacement and St. Jude mitral valve replacement, OmniScience mitral valve replacement, resection of a right ventricular muscle band, placement of a permanent pacemaker, and tricuspid valvotomy (1 each).

A third operation was performed in 6 patients at a mean age of 52 months (range, 6 months to 7 years). These procedures included right ventricular outflow tract enlargement (2 patients), reoperation for coarctation (2), replacement of an apicoaortic conduit, and placement of a left subclavian artery–descending aortic shunt.

Three patients required a fourth operation at 2, 7, and 9 years of age. These procedures included placement of a subclavian artery–descending aortic shunt, pacemaker placement, and reoperation for coarctation.

One patient required a fifth operation, a reoperation for recurrent subaortic stenosis, previously resected when the patient was 2 years of age.

Six patients underwent reoperation for recurrent coarctation. Two of them required shunting from the left subclavian artery to the descending aorta because of inadequate flow from the left ventricle to the descending aorta owing to restriction at the level of the coarctation. No patient had more than one surgical revision of the coarctation, and 2 patients had balloon angioplasty for

Table 1. Summary of Anatomical Data for 30 Patients With Shone's Anomaly

	Lesions in Shone's Anomaly					
Patient No.	Left Atrial Ring	Mitral Valve Abnormality	Subaortic Obstruction	Coarctation	All Four	Other Anomalies
1	No	Stenosis	No	Yes	No	VSD, PDA
2	No	Normal	Yes	Yes	No	PDA, PFO, Bi Ao
3	Yes	Fused chordae	Yes	Yes	Yes	VSD, Bi Ao
4	Yes	Stenosis	No	Yes	No	VSD, PDA, PFO, ASD, hypoplastic arch, Bi Ao
5	Yes	Stenosis	No	Yes	No	None
6	Yes	All to 1 papillary	Yes	Yes	Yes	VSD, PDA, PFO, ASD, hypoplastic left ventricle
7	No	Fused chordae	Yes	No	No	Bi Ao
8	Yes	Stenosis	Yes	Yes	Yes	VSD, Bi Ao
9	Yes	Stenosis	Yes	Yes	Yes	VSD, Bi Ao
10	Yes	Fused chordae	Yes	Yes	Yes	VSD, PDA, Bi Ao
11	No	Normal	Yes	Yes	No	VSD, Bi Ao
12	No	Normal	Yes	Yes	No	VSD, Bi Ao
13	Yes	Parachute mitral	Yes	Yes	Yes	PDA, Bi Ao
14	No	Stenosis/regurgitation	Yes	Yes	No	VSD, PDA, PFO, Bi Ao
15	Yes	Stenosis	Yes	Yes	Yes	PDA
16	No	Parachute mitral	Yes	Yes	No	None
17	Yes	All to 1 papillary	Yes	Yes	Yes	VSD, Bi Ao
18	Yes	Stenosis	Yes	Yes	Yes	VSD, PDA, PFO, ASD
19	Yes	Stenosis	Yes	Yes	Yes	VSD, PDA
20	Yes	Stenosis	Yes	Yes	Yes	None
21	Yes	Stenosis	Yes	Yes	Yes	PFO, Bi Ao
22	Yes	Fused chordae	No	Yes	No	VSD, Bi Ao
23	Yes	All to 1 papillary	Yes	Yes	Yes	Bi Ao
24	Yes	All to 1 papillary	Yes	Yes	Yes	VSD, PDA, PFO
25	Yes	Fused chordae	Yes	Yes	Yes	VSD, PDA
26	Yes	All to 1 papillary	Yes	Yes	Yes	VSD
27	Yes	Parachute mitral	Yes	Yes	Yes	Bi Ao
28	Yes	Stenosis	Yes	Yes	Yes	VSD, Bi Ao
29	No	Normal	Yes	Yes	No	VSD, Bi Ao
30	Yes	Stenosis	Yes	Yes	Yes	VSD, Bi Ao

ASD = atrial septal defect; Bi Ao = bicuspid aortic valve; PDA = patent ductus arteriosus; PFO = patent foramen ovale; VSD = ventricular septal defect.

this problem. Five patients with coarctation underwent attempts at balloon angioplasty that were considered unsuccessful.

Two patients underwent reoperation for recurrent subaortic obstruction that had previously been resected, and 1 patient underwent mitral valve replacement 1 month after a failed mitral commissurotomy.

In total, 84 operative procedures were performed in 28 patients with an average of 2.8 separate procedures per patient occurring at an average of 2.4 operations per patient.

Results

Mortality

There were no operative deaths associated with first operations. All four deaths occurred in the hospital after the second operation (4/17). The first operative death was attributed to low cardiac output after Hancock bioprosthetic valve placement in the mitral position and VSD closure in a child who remained in severe CHF 1 month after a coarctation repair and PDA ligation. This child was 3 months old at the time of death.

The second postoperative death occurred in a 3-year-old child after coarctation repair and a systemic–pulmonary artery shunting procedure, done 2 weeks after an atrial septostomy and mitral valvotomy. The third postoperative death was in a 4-year-old girl who underwent a Konno aortoventriculoplasty with St. Jude prosthetic valve placement in both the aortic and mitral positions. This patient had had a previous flap angioplasty repair of coarctation immediately after birth and remained in severe CHF despite medications. Postopera-

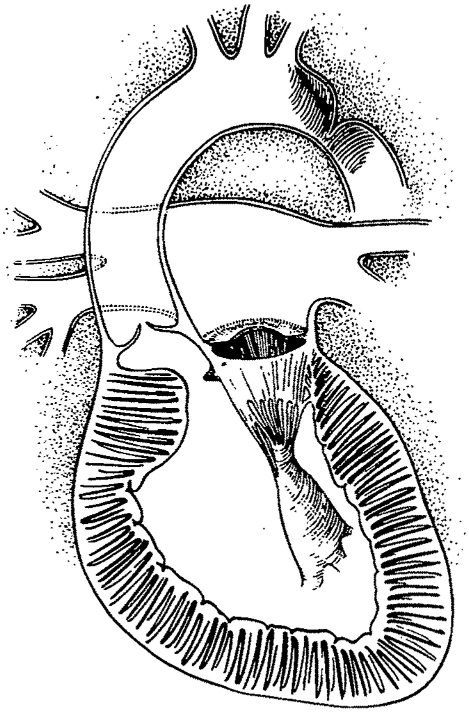

Fig 1. The four obstructive anomalies forming Shone's anomaly: supravalvar ring of the left atrium, parachute mitral valve, subaortic stenosis, and coarctation of the aorta, in that order according to the direction of blood flow. (Reprinted from [1] by permission of the American Journal of Cardiology.)

tive death was attributed to low cardiac output from right coronary artery occlusion.

The fourth patient who died had phocomelia and floppy baby syndrome and was returned to the hospital at age 23 months for a second operation. The second operation included closure of an atrial septal defect and unbanding of the pulmonary artery. At that time, the patient had a restrictive VSD with suprasystemic right ventricular pressures and severe desaturation. Aplasia of the bone marrow and severe thrombocytopenia developed and ultimately led to pulmonary hemorrhage and death. Three of 4 patients who died had all four anatomical anomalies of Shone's. There have been no late deaths.

The aortic cross-clamp time for the first operation requiring cardiopulmonary bypass was not significantly different between survivors and nonsurvivors (23 ± 8 minutes versus 40 ± 5 minutes, respectively).

When catheterization data of operative survivors were compared with those of nonsurvivors, the only significant differences were for pulmonary artery systolic and diastolic pressures. The pulmonary artery systolic pressures were 62 ± 2 mm Hg versus 102 ± 14 mm Hg and pulmonary artery diastolic pressures, 22 ± 3 mm Hg versus 39 ± 9 mm Hg for survivors and nonsurvivors, respectively (both, $p < 0.05$).

Morbidity

There was one serious postoperative complication resulting in a prolonged hospital course after the first operation. It was a sternal wound infection, which resolved with intravenous antibiotics and secondary closure. No organisms were ever identified.

In addition, 1 patient was transferred to our institution with the symptoms of peritonitis and guaiac-positive stools. This patient had all four abnormalities of Shone's and was in florid CHF at the time of presentation. Necrotizing enterocolitis developed after the cardiac operation, and the patient required prolonged hyperalimentation and hospitalization. No abdominal procedure was necessary as a result of this complication.

The incidence of major morbidity increased with each succeeding operation. After the second operation, 1 patient sustained a series of postoperative complications, which included a posterior myocardial infarction, reexploration for bleeding, an upper gastrointestinal bleed, stroke, and deficiency of coagulation factors II and V. This patient had replacement of the apicoaortic conduit and enlargement of the right ventricular outflow tract and is now doing well 19 months after operation. A series of conduction abnormalities due to complete heart block were noted in 1 patient after operation and resulted in placement of a permanent pacemaker. One patient each had right bundle-branch block, right ventricular conduction delay, and left bundle-branch block, but these conditions did not necessitate placement of a permanent pacemaker.

Follow-up Data

Of the 30 patients identified with Shone's anomaly, 4 patients died and no follow-up was available for 1 patient. Of the 2 patients treated medically, 1 returned to Japan after the diagnosis of Shone's anomaly was made, and she was lost to follow-up. This young Oriental girl had adrenogenital syndrome as well as Shone's anomaly. The other medically treated patient had Turner's syndrome as well as mild Shone's anomaly, and is now working as a secretary.

Of the 28 patients who underwent surgical intervention, 24 were eligible for follow-up after their last operative procedure. The average length of follow-up is 5.7 ± 1.1 years after the most recent operation. The latest New York Heart Association activity level was class I for 82%, class II for 12%, and class III for 6%. Seven patients have persistent cardiomegaly on chest roentgenograms. Six patients still require medical therapy for control of persistent episodes of CHF.

At the most recent postoperative catheterization, the mean follow-up gradient through the area of repaired aortic coarctation was 16 ± 4 mm Hg (7 patients) and across the aortic outflow tract, 33 ± 10 mm Hg (5 patients). Two patients still have apicoaortic conduits in place.

Only 4 patients were reported to have a residual gradient across the mitral valve (mean gradient, 5 ± 1 mm Hg). Other reported valvar abnormalities at follow-up were mild mitral regurgitation in 3 patients and moderate tricuspid regurgitation in 1 patient.

Although these patients with Shone's anomaly remain quite small physically, success in school was at least average in this group based on follow-up reports. Furthermore, only 3 of the patients in the follow-up group were

Table 2. Summary of Operative Procedures From Most Recent to Initial

Patient No.	Operative Repairs and Procedures
1	1: Redo CoA repair with Dacron; 2: VSD closure; 3: CoA repair and PDA ligation
2	1: CoA repair with Gore-Tex patch and PDA ligation
3	1: Mitral valvotomy; 2: CoA balloon angioplasty for restenosis; 3: CoA repair
4	1: Redo CoA repair with Dacron; 2: CoA repair
5	1: CoA repair with LSCA patch
6	No repair
7	1: Open aortic and mitral commissurotomies
8	1: CoA repair (end to end)
9	1: Subaortic resection of muscle band, VSD closure; 2: CoA repair
10	1: Aortic valvotomy, LV outflow tract myomectomy, VSD closure; 2: subaortic membrane excision; 3: PDA ligation, CoA repair
11	1: Resection of subaortic restenosis; 2: redo CoA repair; 3: resection and patch of RV outflow tract obstruction; 4: VSD closure; 5: PDA ligation, CoA repair
12	1: CoA repair and PDA ligation
13	1: CoA repair and PDA ligation
14	1: Hancock bioprosthetic replacement of mitral valve, VSD closure; 2: CoA repair, PDA ligation
15	1: CoA repair, Potts shunt; 2: atrial septostomy, mitral valvotomy
16	No repair
17	1: CoA repair with LSCA
18	1: PA banding, CoA repair; 2: right Blalock-Taussig shunt, atrial septostomy
19	1: VSD closure; 2: CoA repair with LSCA, PDA ligation
20	1: RV outflow tract enlargement, replacement of apicoaortic conduit; 2: LV apex–aorta conduit; 3: CoA repair
21	1: Mitral valvotomy; 2: CoA repair
22	1: Redo CoA repair with Dacron; 2: CoA repair with LSCA
23	1: CoA repair with LSCA; 2: aortic commissurotomy, resection of subaortic stenosis
24	1: CoA repair with Gore-Tex, PDA ligation
25	1: Konno aortoventriculoplasty with St. Jude replacement of aortic and mitral valves, CoA balloon angioplasty for restenosis; 2: CoA repair with LSCA
26	1: PA banding, CoA repair
27	1: CoA repair (end to end)
28	1: LSCA–descending aortic shunt; 2: CoA repair (end to end); 3: MVR with No. 17 Omniscience; 4: mitral commissurotomy
29	1: Pacemaker placement for third-degree heart block; 2: resection of subaortic band, resection of RV band, patch closure of VSD; 3: Ao valvotomy, tricuspid valvotomy, PDA ligation; 4: CoA repair with LSCA
30	1: PDA, CoA repair with LSCA; 2: Closure of VSD

CoA = coarctation of aorta; LSCA = left subclavian artery; LV = left ventricular; MVR = mitral valve replacement; PA = pulmonary artery; PDA = patent ductus arteriosus; RV = right ventricular; VSD = ventricular septal defect.

considered to be "minimally" handicapped or to have learning disabilities.

Comment

More than 25 years have passed since Shone and colleagues [1] first described the developmental complex of parachute mitral valve, supravalvar ring of the left atrium, subaortic stenosis, and coarctation of the aorta. In that original article (8 patients), only 2 patients exhibited all four aspects of Shone's anomaly and could therefore be considered to have pure cases of Shone's anomaly. In our series, 19 patients were seen with all four aspects of the anomaly and therefore were considered to have pure cases. The other 11 can be regarded as having forme fruste of Shone's anomaly, most missing a supravalvar mitral ring.

In the description of Shone and associates [1], it was noted that mitral valve obstruction appeared to be the most critical problem associated with the anomaly, and mitral commissurotomy alone (performed in 3 of the patients) was not adequate to relieve the mitral valve obstruction. Shone and colleagues quite farsightedly made a hypothetical case for excision of the supravalvar ring and replacement of the mitral valve with a prosthesis as the best method of overcoming the major mitral valvar and subvalvar obstructions.

Our present study confirms the belief that severity of the mitral valve obstruction correlates inversely with long-term outcome and that operative mortality in patients with Shone's anomaly is adversely affected by the degree of mitral valve disease. The patients who demonstrated substantially elevated pulmonary artery pressures were those with the worst mitral obstruction and had, in general, the poorest outcome.

These patients also appeared to have severe and progressive left ventricular outflow tract obstruction. Kieffer and associates [2] noted an association between reversed ventricular septal curvature seen on two-dimensional echocardiography with primary pulmonary hypertension and Shone's anomaly. Indeed, our patients with severe mitral obstructive disease as a result of Shone's anomaly did have secondary pulmonary hypertension, which conceivably could have led to increased reversed ventricular septal curvature and exacerbation of subaortic obstruction.

Rosenquist [3] described the results of pathological study of 53 heart specimens in which the primary diagnosis was coarctation of the aorta with normally related great vessels. Rosenquist noted a surprisingly high incidence of obstructive mitral valve involvement in these specimens. Furthermore, the mitral valve abnormalities usually involved the subvalvar apparatus and demonstrated a wide spectrum of anomalies from hypoplastic but otherwise normal mitral valves to severe parachute mitral stenosis.

In addition, that report [3] noted three abnormalities in the mitral valves of patients with Shone's anomaly, findings subsequently confirmed by others [4]. These abnormalities involved the mitral subvalvar apparatus and included fusion of the papillary muscles, fusion of a papillary muscle to the ventricular wall, and true parachute mitral valve with only one papillary muscle. Varying degrees of all these forms of subvalvar mitral abnormality were noted in our patients (see Table 1).

In a clinicopathological conference report demonstrating the case of a patient with Shone's anomaly [5], physicians were cautioned to exercise an awareness of the possibility of other left-sided obstructive lesions that could be present in the symptomatic infant with coarctation. The correlation of obstructive left-sided anomalies associated with coarctation of the aorta was confirmed by Becker and co-workers [6] 2 years later. They believed that Shone's anomaly should be excluded in those patients who continue to have signs of CHF after operative repair of the coarctation. This correlates very well with our present group of patients, many of whom demonstrated continued CHF after the first operation, which was most commonly coarctation repair and ligation of patent ductus arteriosus.

As recently as 1987 in a report of spontaneous complete atrioventricular conduction block in Shone's anomaly [7], it was stated that the diagnosis of Shone's anomaly is usually made during infancy or early childhood. However, the authors then noted that most patients with Shone's anomaly are dead soon after the diagnosis is made or soon after an attempt at surgical repair.

Many cardiac surgeons and cardiologists agree with this dismal outlook, and therefore, the prevailing sentiment is that the prognosis for these patients is poor. However, few data are available to document the outcome of patients with Shone's anomaly. Although there have been isolated case reports [8] describing the operative approach to the different lesions that make up Shone's anomaly, no overall operative strategy has been developed. Our operative approach to these patients includes early repair of coarctation and ligation of patent ductus arteriosus. Repair of other lesions including VSD can generally be postponed. The majority of VSDs in our series were restrictive and closed spontaneously after coarctation repair.

The surgical options for the left ventricular outflow tract obstruction must be tailored to the individual patient because of the wide variability in anatomy. When the aortic annulus is adequate and the obstruction is limited to the subaortic area, septoplasty is an effective procedure. If the annulus is hypoplastic, we favor aortoventriculoplasty (Konno procedure) [9, 10] over left ventricular apex–descending aortic shunts. Aortoventriculoplasty has been used for tunnel subaortic obstruction, and the incision in the interventricular septum can easily be taken well into the left ventricular cavity, effectively relieving the obstruction. The Konno procedure allows the aortic valve to remain in the normal anatomical position with normal flow characteristics and permits the placement of adequately large valves, despite the overall small body size of the patient. This procedure has many other advantages compared with apicoaortic conduit procedures, including decreased arrhythmias and preservation of ventricular function [11].

The operative treatment of the mitral lesions noted in these patients remains difficult because of the highly variable nature of these malformations and the differing degrees of subvalvar involvement. However, valve repair should be considered whenever feasible in these young patients before mitral valve replacement is performed.

The frequency of multiple operations in patients with Shone's anomaly (18 of 30 patients undergoing more than one procedure) makes this group a formidable technical challenge. Furthermore, the high risk of subsequent operations, particularly in those patients with mitral valve involvement, can discourage even the most aggressive surgeon. Finally, although our patients have had a satisfactory long-term outcome, 4 of 24 children have a prosthetic valve in place and must undergo further reoperative procedures to replace bioprosthetic valves as they degenerate or suffer the consequences of anticoagulation, which have led to substantial gastrointestinal bleeding in 2 patients.

In summary, our report demonstrates that patients with Shone's anomaly represent a wide spectrum of major anatomical and hemodynamic abnormalities. An aggressive surgical approach tailored to each patient's individual anatomy can result in a satisfactory outcome for most of these patients.

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DISCUSSION

DR KEVIN TURLEY (San Francisco, CA): I congratulate Bolling and associates for the presentation and the excellent results in this difficult group of patients.

As noted, since the first description of the developmental complex of parachute mitral valve, supravalvar ring of the left atrium, subaortic stenosis, and coarctation of the aorta by John Shone and associates in 1963, relatively little has been written about the natural history of the patients who had surgical intervention. However, quite a bit has been contributed to the literature in recent years on the problems of mitral valve abnormalities, especially in the neonate. I think critical to the discussion of this report is an understanding of the patient population and the problems associated with this complex.

In the group presented, only 19 patients, 63%, had the full Shone's syndrome, and in the manuscript it is noted that 7 patients actually had operations on the mitral valve—three initial valvotomies, two second-operation valvotomies, placement of one bioprosthesis, and placement of one mechanical valve. Among those 7 patients, there were four deaths. All four deaths happened at the second operation. These patients, as noted, were representative of the pure Shone's complex.

There were 22 patients with supravalvar mitral rings and 26 patients with mitral valve abnormalities, including parachute mitral valve, fused chordae, and the single papillary muscle. However, only 7 patients had operations on the mitral valve. I think we would like to know more about the other 19 patients with mitral valve abnormalities and the 17 with supravalvar rings and what happened to these valve structures over time.

Further, coarctation was a major problem in this series, as it is for all patients with this syndrome, and 3 of the patients finally received left subclavian–descending aortic grafts. Dr Bolling, could you tell us if there was anything special about the problems of these patients at reoperation for coarctation that necessitated this approach?

I agree with the conclusion of Bolling and associates that critical to this problem is the degree of mitral valve disease. I think with both an aggressive surgical approach, as they demonstrated, tailored to the other lesions, and improved techniques of mitral reconstruction, gratifying results can be achieved.

DR BOLLING: The critical point to be noted in patients with Shone's anomaly is the degree of mitral valve obstruction. Less than half of these patients had mitral valve procedures, and that is one of the problems with a historical review. I think we would agree completely with Dr Turley that our approach to the mitral valve in patients operated on now would be more aggressive. It is interesting to note that in the initial article by Shone and co-workers, which was an autopsy series of 8 patients, 3 of the patients had had previous mitral commissurotomies, and that the authors thought that mitral commissurotomy was not sufficient to repair the mitral disease.

In response to the question about left subclavian–descending aortic bypass grafting, these patients are in a historical group, and patients seen more recently would have coarctation repair by other methods. Those patients were, of course, in the group with recurrent coarctation.

Postischemic [Ca^{2+}] Repletion Improves Cardiac Performance Without Altering Oxygen Demands

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The positive inotropism expected with correction of postischemic hypocalcemia might be counterbalanced by potential aggravation of reperfusion injury, in particular by calcium overload. We evaluated the effect of normalizing blood calcium concentration ([Ca^{2+}]) on postischemic left ventricular systolic and diastolic mechanics using oxygen consumption and indices derived from pressure–diameter relations. In 10 open-chest dogs on cardiopulmonary bypass, the hearts underwent 30 minutes of normothermic global ischemia followed by one hour of multidose hypothermic (4°C), hypocalcemic (0.3 mmol/L) blood cardioplegia. After reperfusion, systemic [Ca^{2+}] had decreased to 70% of control ($p = 0.017$). The left ventricular inotropic state was significantly depressed from baseline (control) values, but was restored to baseline levels by resumption of normocalcemia after one hour of reperfusion. Chamber stiffness increased by 308% ($p = 0.006$) after hypocalcemic reperfusion but decreased significantly after [Ca^{2+}] correction. Recovery of left ventricular performance with [Ca^{2+}] correction did not augment myocardial oxygen consumption from the postischemic uncorrected state (5.0 ± 0.3 mL O_2/min/100 g versus 5.3 ± 0.3 mL O_2/min/100 g). We conclude that normalizing [Ca^{2+}] after blood cardioplegia improves postischemic left ventricular performance without adversely affecting compliance or oxygen consumption.

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Hypothermic blood cardioplegia (BCP) is widely used during elective open-heart operations. The formulation of BCP is based on the synergistic concepts of reducing oxygen demands to prevent ischemic injury and of modifying the conditions and composition of the perfusate to prevent reperfusion injury [1, 2]. An important strategy in the modification of myocardial reperfusate is to decrease calcium influx with chelating agents or calcium-channel blockers in the cardioplegic solution to avoid calcium-related injury during reperfusion [1, 3–5]. One consequence of calcium chelators in BCP concomitant with hemodilution during cardiopulmonary bypass is an acute reduction in systemic ionized calcium (Ca^{2+}) levels. Normalization of plasma Ca^{2+} levels during the early phase of reperfusion could potentially improve myocardial performance, but could also aggravate postischemic calcium overload, inappropriately augment inotropism and left ventricular (LV) afterload [6], and increase oxygen demands during a metabolically vulnerable period.

This study was designed to determine whether restoration of postischemic plasma Ca^{2+} levels is beneficial or deleterious to postischemic LV performance and oxygen demands. We evaluated the changes in myocardial oxygen consumption per minute (MV_{O_2}) and in LV systolic and diastolic performance before and after the administration of calcium chloride ($CaCl_2$) to normalize systemic blood Ca^{2+} levels in canine hearts subjected to 30 minutes of normothermic ischemia followed by cardioplegic arrest. Left ventricular systolic performance was assessed using linear indices based on instantaneous pressure–dimension relations. Chamber stiffness was evaluated by the polynominal end-diastolic pressure–dimension relations.

Material and Methods

Surgical Procedure

Ten heartworm-free adult dogs weighing 20.5 to 30 kg were anesthetized with sodium pentobarbital, 30 mg/kg intravenously, supplemented by a continuous infusion of 2 to 4 mg/kg/h to maintain a constant level of anesthesia. Each dog was intubated and ventilated with oxygen-enriched room air by a volume-cycled respirator. A standard lead II electrocardiogram was monitored, and polyethylene catheters were inserted in the femoral artery and vein for arterial blood sampling and fluid or drug administration, respectively.

The heart was exposed through a median sternotomy, and the azygos vein was ligated. The pericardium was incised and tented to cradle the heart. Millar MPC-500 solid-state transducer-tipped catheters were placed in the proximal aorta through the right internal mammary artery and in the left ventricle through an apical stab wound to measure instantaneous aortic and LV pressures, respectively. After heparinization (3 mg/kg, supplemented every 1.5 hours), the superior and inferior venae cavae were

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transatrially cannulated and snared with umbilical tapes, and the dog was placed on total cardiopulmonary bypass.

Systemic venous blood was directed through a bubble oxygenator (model S100A; Shiley Inc, Irvine, CA) and returned to the dog through a cannula in the left subclavian artery using a bidirectional roller pump. Oxygen flow rate was adjusted to maintain arterial oxygen tension greater than 150 mm Hg and arterial carbon dioxide tension between 30 and 40 mm Hg. Sodium bicarbonate was administered as necessary to maintain pH between 7.38 and 7.42. Systemic perfusion pressure was maintained at 80 mm Hg by adjusting the subclavian flow rate. Single-donor homologous blood and Hespan (6% hetastarch in 0.9% sodium chloride; DuPont Pharmaceuticals, Wilmington, DE) were used to prime the oxygenator reservoir, with the final hematocrit being greater than 0.20. Methylprednisolone (Solu-Medrol; Upjohn Co, Kalamazoo, MI), 1,000 mg, was added to minimize pulmonary capillary fragility and transcompartmental fluid migration during cardiopulmonary bypass.

Right heart bypass was then established to control LV preload by infusing oxygenated blood through a cannula introduced into the main pulmonary artery through the infundibulum of the right ventricle. The left and right ventricles were vented by direct cannulation and gravity drainage. Total coronary blood flow (CBF) was measured by direct collection of coronary venous effluent through the right ventricular vent. For the anaerobic collection of venous blood, a special glass bottle was devised in which nitrogen gas continuously purged the air space above the collected coronary venous blood. This system allowed integration of the blood sample during the delivery period of BCP.

Two ultrasonic (5-MHz) crystals 3 mm in diameter were placed subendocardially in the LV free wall, one in the anterior wall and one in the posterior wall, to measure instantaneous minor-axis diameter with a sonomicrometer (model 120; Triton Technology, San Diego, CA). A double-lumen catheter (DLP Inc, Grand Rapids, MI) was secured in the ascending aorta for delivery of BCP and simultaneous measurement of infusion pressure. A gauze pad soaked in 40% formaldehyde was applied topically to the tissue around the junction of the superior vena cava and the right atrium to inactivate the intrinsic pacemaker activity of the sinoatrial node, and atrial pacing was established at a rate of 150 beats per minute by an external pacemaker (model 5880A; Medtronic, Inc, Minneapolis, MN). The pericardial sac was subsequently lavaged with warm saline solution to remove the formaldehyde. A Shiley thermistor probe was placed in the interventricular septum to monitor myocardial temperature.

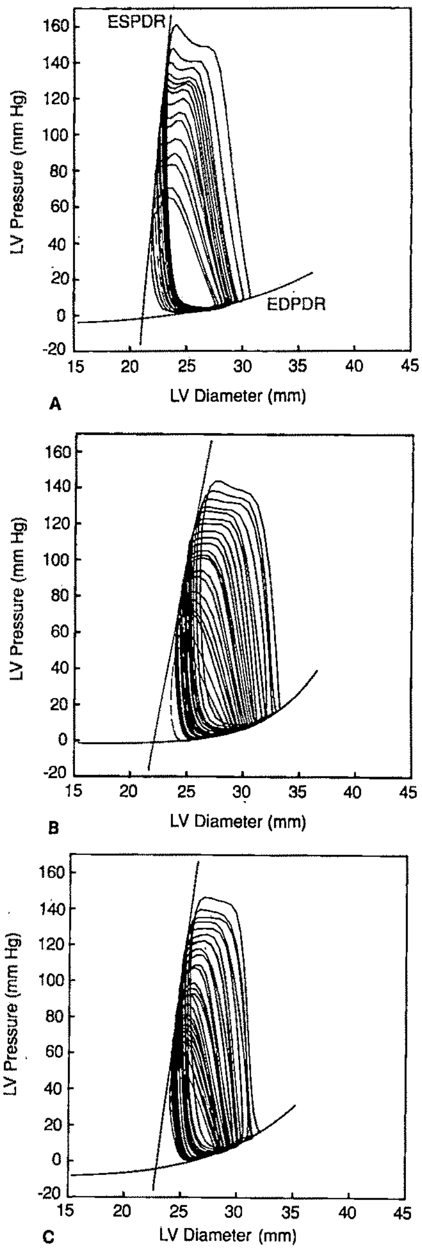

Fig 1. Representative left ventricular (LV) *pressure–diameter loops generated by afterload reduction during (A) control, (B) postischemic hypocalcemic state, and (C) postischemic normocalcemic state. Linear regression lines show end-systolic pressure–dimension relations* (ESPDR) *and polynomial curves show end-diastolic pressure–dimension relations* (EDPDR).

Measurements and Calculations

Data were displayed on a multichannel 8890 recording and monitoring system (Hewlett-Packard, Andover, MA), and instantaneous LV pressure–dimension loops were displayed on-line by a dual-beam X-Y oscilloscope (model 502A; Tektronix, Portland, OR). Transient reduction of LV afterload was employed to generate a series of variably loaded pressure–dimension loops, as shown in Figure 1. To accomplish this maneuver, cardiac output was maintained at 80 mL/min/kg body weight by right heart bypass, and mean arterial pressure was gradually reduced from 140 to 50 mm Hg over 15 seconds by adjusting the subclavian roller pump. Data were recorded on both analog frequency modulation magnetic tape (Hewlett-Packard 3968A) and a microcomputer system (IBM PC-AT) using an analog-digital converter (model DT2821; Data Translation, Marlborough, MA) sampling at 200 Hz. The digitized data were processed by computer algorithms using an interactive videographics program designed in our laboratory. End-diastolic points were iden-

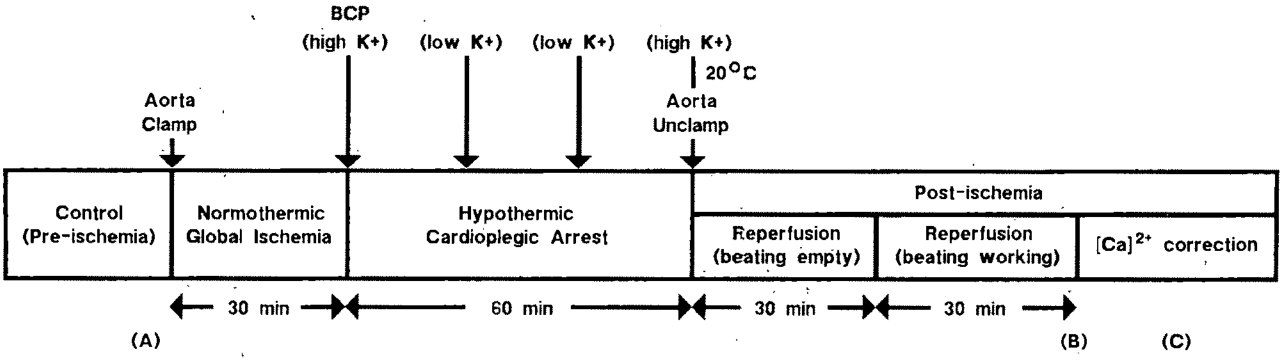

Fig 2. The protocol. Hearts placed on cardiopulmonary bypass were subjected to 30 minutes of global ischemia followed by 60 minutes of arrest using intermittent (20 minutes) hypothermic (4°C), hypocalcemic (0.3 mmol/L) blood cardioplegia (BCP). The hypocalcemic state (L-[Ca^{2+}]) was sustained during one hour of normothermic reperfusion, and then [Ca^{2+}] was corrected to the value equivalent to baseline values (N-[Ca^{2+}]). (A), (B), and (C) represent the time of data acquisition at control, postischemic L-[Ca^{2+}] state, and postischemic N-[Ca^{2+}] state, respectively.

tified as the relative minima after atrial contraction on the LV pressure trace, and were adjusted manually if necessary using the interactive videographics program.

END-SYSTOLIC PRESSURE–DIMENSION RELATION. The end-systolic pressure (P_{es})–diameter (D_{es}) points were identified as the upper left corner of the pressure–diameter loops using the iterative technique of Kono and colleagues [7]. The P_{es}–D_{es} points were fitted to the linear equation

$$P_{es} = M_{es}(D_{es} - D_{es,0}) \qquad \text{(equation 1)}$$

where M_{es} is the maximal elastance (slope of the P_{es}–D_{es} relation) and $D_{es,0}$ is the diameter intercept at P_{es} = 0 mm Hg [8].

PRELOAD RECRUITABLE DIMENSIONAL STROKE WORK. Dimensional stroke work (SW) was calculated using point-by-point integration of LV pressure and diameter ($SW = \int P \cdot dD$) [9]. Regression analysis was performed to fit dimensional SW to corresponding end-diastolic diameters (D_{ed}):

$$SW = M_{SW}(D_{ed} - D_{SW,0}) \qquad \text{(equation 2)}$$

where M_{SW} is the slope of the linear SW–D_{ed} relation, and $D_{SW,0}$ is the diameter intercept where SW = 0 mm Hg · mm.

Left ventricular performance was quantitated by the slope and diameter axis intercept. In addition, the positions of the end-systolic pressure–dimension relation and preload recruitable SW are described using the midrange diameter values calculated from the median value at a P_{es} of 100 mm Hg ($D_{es,100}$) and an SW of 400 mm Hg · mm ($D_{SW,400}$) [10], which integrate changes in both slope and diameter axis intercept.

END-DIASTOLIC PRESSURE–DIMENSION RELATION. The variably loaded end-diastolic pressure (P_{ed})–diameter (D_{ed}) points were also used to determine the characteristics of LV chamber stiffness (see Fig 1). The P_{ed}–D_{ed} points in each loop were fitted to a formula:

$$P_{ed} = \alpha[(D_{ed}/D_0)^{\beta} - 1] \qquad \text{(equation 3)}$$

where α indicates the extrapolated P_{ed} at D_{ed} = 0 mm, β indicates the intensity of the curvature indicative of a modulus of LV chamber stiffness, and D_0 is the dimension axis intercept at zero P_{ed}.

OXYGEN CONSUMPTION. Blood samples were drawn from the arterial infusion line and the right ventricular collection system to measure, respectively, arterial oxygen content (CaO_2) and coronary venous oxygen content (CvO_2) (co-oximeter model 282; Instrumentation Laboratory, Lexington, MA). Myocardial oxygen extraction (Eo_2) as a percentage of $M\dot{V}o_2$ in milliliters of oxygen per minute per 100 g were calculated as follows:

$$Eo_2 = [(CaO_2 - CvO_2)/CaO_2](100)$$

and

$$M\dot{V}o_2 = [(CaO_2 - CvO_2) \times CBF](100/HWT)$$

where CBF is measured in milliliters per minute from the right ventricular vent and HWT = heart weight in grams.

Experimental Protocol

The experimental protocol was designed to be relevant to the clinical setting of surgical reperfusion, where inotropic agents may be administered during discontinuation of extracorporeal support. The global (normothermic) ischemia model was chosen because it creates a predictable degree of LV depression as well as sensitivity to calcium during reperfusion. This model is relevant to the clinical counterpart of poor ventricular performance preoperatively as well as multivessel coronary artery disease or large acute myocardial infarctions. A schematic time course of the protocol is presented in Figure 2.

CONTROL. The condition of each dog was allowed to stabilize on total vented bypass for 15 minutes after surgical preparation. Blood [Ca^{2+}] was measured using a thermostatically controlled flow-through calcium-selective electrode system (NOVA Biomedical, Waltham, MA) and was maintained at 1.0 mmol/L using a drip infusion of $CaCl_2$ (0.1 to 0.2 mmol/L/min) into the oxygenator reservoir. With mean arterial pressure adjusted to

Table 1. Composition of Blood Cardioplegia

Constituent	Concentration
K^+ (KCl) (mmol/L)	20–25 (high $[K^+]$) 10 (low $[K^+]$)
Ca^{2+} (CPD) (mmol/L)	0.3
Mg^{2+} (derived from blood) (mmol/L)	0.5
pH (THAM buffer)	8.20 (4°C)
Osmolality (mosm/L)	360–380
Hematocrit	0.14–0.18
Oxygen content (mL/O_2/100 mL)	8–10

CPD = citrate-phosphate-dextrose (blood); THAM = trihydroxymethylaminomethane.

80 mm Hg, the baseline (control) oxygen metabolic data were obtained during both the beating empty (BE) and the beating working (BW) states, the latter while cardiac output was controlled at 80 mL/min/kg body weight. Ventricular performance was assessed using the previously described afterload reduction procedure repeated three to five times.

NORMOTHERMIC GLOBAL ISCHEMIA. The heart was then returned to the vented BE state, and the aorta was cross-clamped for a total of 30 minutes of normothermic global ischemia. Myocardial temperature ranged from 33° to 35°C.

HYPOTHERMIC BLOOD CARDIOPLEGIA. After global ischemia, the first infusion of 4°C BCP (Table 1) was delivered at 50 mm Hg perfusion pressure for three minutes in a ratio of 4 parts blood to 1 part crystalloid [2]. Iced saline slush was applied topically for myocardial cooling, and each dog underwent moderate systemic hypothermia (28°C). The BCP with a potassium concentration ($[K^+]$) of 10 mmol/L (low $[K^+]$; see Table 1) was replenished intermittently every 20 minutes by a two-minute infusion. After one hour of hypothermic cardioplegic arrest, systemic blood temperature was rewarmed to 37°C and a moderately warm BCP (20°C) was delivered into the aortic root for three minutes, while $[K^+]$ was increased to 20 mmol/L (high $[K^+]$; see Table 1) to avoid resumption of electromechanical activity at this warmer temperature. Coronary venous effluent was collected anaerobically to measure $M\dot{V}O_2$ during each infusion of BCP. The mean systemic perfusion pressure was maintained at 50 mm Hg during the entire period of hypothermic cardioplegia.

REPERFUSION. The aortic cross-clamp was removed after the last BCP delivery. Blood $[Ca^{2+}]$ was intentionally left in an uncorrected low calcium level (L-$[Ca^{2+}]$). Ventricular fibrillation, when encountered, was immediately converted by direct-current countershock. After electromechanical activity resumed, perfusion pressure was increased to 80 mm Hg over three minutes through the subclavian pump, and the heart was left in the BE state for 30 minutes. The BW state was then established for 30 minutes at a cardiac output of 80 mL/min/kg and a mean arterial pressure of 80 mm Hg, after which postischemic metabolic values were obtained in both the BE and the BW states, and cardiac performance was measured.

CALCIUM CORRECTION. With the heart in the BW state, $[Ca^{2+}]$ in the pump reservoir was subsequently corrected to control values by a bolus and a supplemental drip infusion of $CaCl_2$ to avoid rapid fluctuations in $[Ca^{2+}]$ and to obtain a steady normocalcemic concentration of 1 mmol/L (N-$[Ca^{2+}]$). Data collections of oxygen metabolism in each hemodynamic state (BE and BW) and LV performance were repeated. The heart was then arrested with a bolus injection of potassium chloride, excised, trimmed of adipose tissue and great vessels, and weighed.

All animals received humane care in compliance with the "Guide for the Care and Use of Laboratory Animals" published by the National Institutes of Health (NIH publication No. 85-23, revised 1985). The protocol was approved by the institutional animal care and use committee.

Statistical Analysis

All data were analyzed with the Statistical Analysis System program (PC-SAS; SAS Institute, Cary, NC) and are presented as the mean ± the standard error of the mean. The multivariate analysis of variance for repeated measures was employed, followed by the Wilks λ test to locate the sources of differences in each sequential study point. Chamber stiffness coefficients were analyzed by covariance analysis using the α and β terms as covariates. Statistical significances were corrected for multiple comparisons using Holm's multiple rejective method for an overall probability of less than 0.05.

Results

Gas tensions and pH of arterial perfusate were comparable at each study point. The $[Ca^{2+}]$ decreased by 30%, from 1.00 ± 0.02 mmol/L to 0.70 ± 0.02 mmol/L during the L-$[Ca^{2+}]$ postischemic period ($p = 0.017$), presumably secondary to hemodilution and admixture of citrate from the BCP solution. It was subsequently restored to control levels (0.95 ± 0.03 mmol/L) after calcium correction.

End-Systolic Pressure–Dimension Relation

The P_{es}–D_{es} points and regression data from the experiment used in Figure 1 are shown in Figure 3A. The P_{es}–D_{es} points are distributed over a wide range (93.6 ± 3.2 mm Hg in P_{es}) and are closely approximated by the linear relationship with little variability around the regression line ($r = 0.9793 \pm 0.0034$). In the postischemic L-$[Ca^{2+}]$ state, the end-systolic pressure–dimension relation showed a generalized rightward and downward shift involving both a decrease in slope and an increase in $D_{es,0}$. Correction of extracorporeal $[Ca^{2+}]$ to normal levels or N-$[Ca^{2+}]$ restored the slope value toward control.

On the average of 10 animals, the slope of the end-systolic pressure–dimension relation (M_{es}) markedly declined after ischemia to 45.8% of control ($p = 0.0006$) (Fig 3B). This decreased slope was accompanied by a significant increase in both $D_{es,0}$ ($p = 0.0039$) (Fig 3C) and $D_{es,100}$

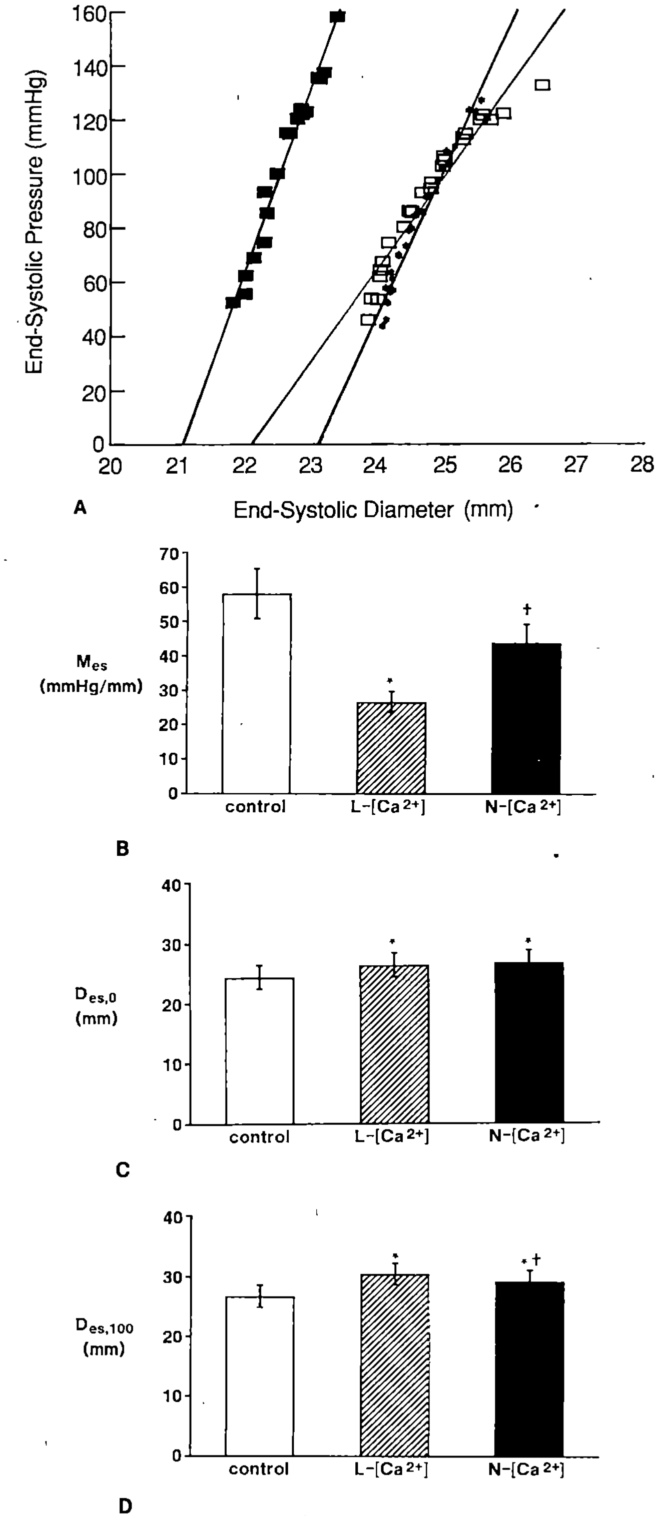

Fig 3. (A) Plots of end-systolic pressure–dimension points determined from the loops shown in Figure 1. (solid squares = control; open squares = *postischemic hypocalcemic or L-[Ca^{2+}] state;* * = *postischemic normocalcemic or N-[Ca^{2+}] state.) (B) The average of the slopes of end-systolic pressure–dimension relation (M_{es}). (C) The diameter axis intercepts ($D_{es,0}$). (D) The midrange diameter intercepts at the end-systolic pressure of 100 mm Hg ($D_{es,100}$). Data are shown as the mean ± the standard error of the mean. (* above bars = significantly different from control; † above bar = significantly different from L-[Ca^{2+}] state.)*

($p = 0.0001$) (Fig 3D), indicating postischemic ventricular dilatation. With [Ca^{2+}] correction, M_{es} was significantly restored to 75.5% of control ($p = 0.0019$ versus L-[Ca^{2+}], which was not significantly different from control M_{es} ($p = 0.0895$). After ischemia, $D_{es,0}$ remained essentially unchanged from L-[Ca^{2+}] ($p = 0.34$), while $D_{es,100}$ showed a small but consistent decrease after normalization of [Ca^{2+}] ($p = 0.0074$), indicating a reduction in the amount of ventricular dilatation in the normal operating range of LV end-systolic pressures.

Preload Recruitable Dimensional Stroke Work

The SW–D_{ed} relation demonstrated a high degree of linearity in all three periods in all experiments ($r = 0.9435 \pm 0.0181$), as Figure 4A illustrates for a representative experiment. In the postischemic state during L-[Ca^{2+}], there was a significant reduction in slope (M_{SW}) to 65.7% of control ($p = 0.0097$) (Fig 4B), with significant increases in both $D_{SW,0}$ ($p = 0.002$) (Fig 4C) and $D_{SW,400}$ ($p = 0.0023$) (Fig 4D). Correction of [Ca^{2+}] significantly increased M_{SW} to 86.9% of control ($p = 0.2948$ versus control). By virtue of this increase in M_{SW}, there was a significant decrease in $D_{SW,400}$ from the L-[Ca^{2+}] level ($p = 0.0110$) without any further change in $D_{SW,0}$ ($p = 0.6893$).

Left Ventricular Chamber Stiffness

Figure 5A shows the end-diastolic curves of each condition using average values of whole experiments computed by the polynomial formula described in equation 3. The α coefficient tended to decrease in the L-[Ca^{2+}] and N-[Ca^{2+}] states, but these were not significant changes. In contrast, the β coefficient in the postischemic L-[Ca^{2+}] state increased threefold over control ($p = 0.0062$) (Fig 5B), with a concomitant rightward shift in D_0 ($p = 0.0038$) (Fig 5C), indicating increased chamber stiffness and dilatation. This is in agreement with both $D_{es,0}$ and $D_{SW,0}$ data. Correction of [Ca^{2+}] substantially decreased the β coefficient ($p = 0.0231$ versus L-[Ca^{2+}]), indicating a decrease in chamber stiffness, but β still remained higher than control ($p = 0.0123$). In addition, D_0 decreased toward control ($p = 0.0582$ versus control) after [Ca^{2+}] correction.

Coronary Blood Flow

As shown in Figure 6A, CBF at control was augmented from the BE state to the BW state ($p < 0.05$) in accordance with the increment in work load. In the postischemic L-[Ca^{2+}] period, there was a pronounced increase in myocardial blood flow in both the BE ($p = 0.0211$) and the BW ($p = 0.0025$) states over the control value. There was no change in CBF with [Ca^{2+}] correction.

Oxygen Consumption

At control, Eo_2 increased with transition from the BE state to the BW state (Fig 6B). This increase in Eo_2, in combination with the increase in CBF, resulted in a significant increase in $M\dot{V}o_2$ (Fig 6C) because of the greater oxygen demands of the working heart. Although the Eo_2 tended to decrease in the postischemic L-[Ca^{2+}] BW state, CBF increased, resulting in an $M\dot{V}o_2$ comparable to control. After [Ca^{2+}] correction, $M\dot{V}o_2$ in the BE state increased significantly ($p = 0.0001$ versus L-[Ca^{2+}]), primarily be-

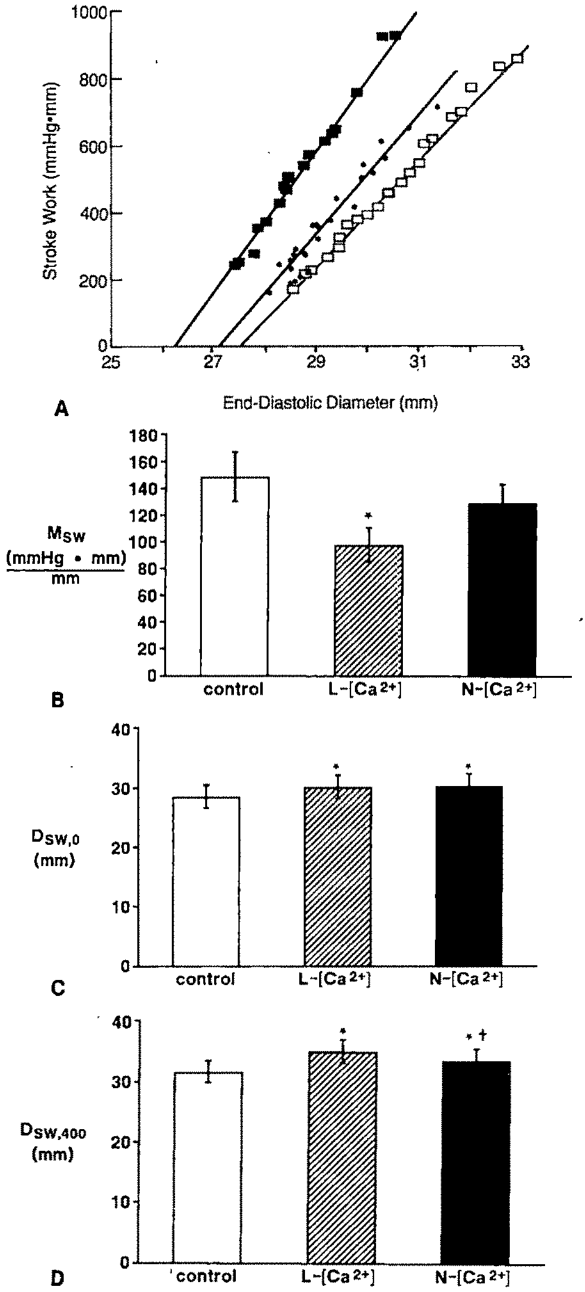

Fig 4. (A) Plots of dimensional stroke work (SW)–end-diastolic dimension points determined from the loops shown in Figure 1. (solid squares = *control;* open squares = *postischemic hypocalcemic or L-[Ca^{2+}] state;* * = *postischemic normocalcemic or N-[Ca^{2+}] state.) (B) The average of the slopes (M_{SW}). (C) The diameter axis intercepts ($D_{SW,0}$). (D) The midrange diameter intercepts at the SW of 400 mm Hg · mm ($D_{SW,400}$). Data are shown as the mean ± the standard error of the mean. (* above bar = significantly different from control; † above bar = significantly different from L-[Ca^{2+}] state.)*

cause of an increase in Eo_2. However, there was no further increase in $M\dot{V}o_2$ in the BW state after normalization of [Ca^{2+}] over that in the L-[Ca^{2+}] period ($p = 0.35$ versus L-[Ca^{2+}]).

Blood Cardioplegia Delivery

The total volume of BCP delivered at the various infusion intervals differed depending on the duration of infusion.

Myocardial oxygen consumption during the BCP infusions corrected for heart weight was highest during the induction period, indicating a substantial uptake and utilization of oxygen despite the 4°C delivery temperature. Myocardial oxygen consumption decreased significantly during the subsequent infusions (Fig 7).

Comment

The management of [Ca^{2+}] in the postischemic heart is a complex problem and has the potential for detrimental as well as beneficial results. The postischemic myocyte is vulnerable to the unbridled entry and accumulation of calcium during the early phase of reperfusion. This accumulation of intracellular calcium has been suspected of initiating or accelerating the transition from reversible to irreversible damage [3–5]. Administration of calcium dur-

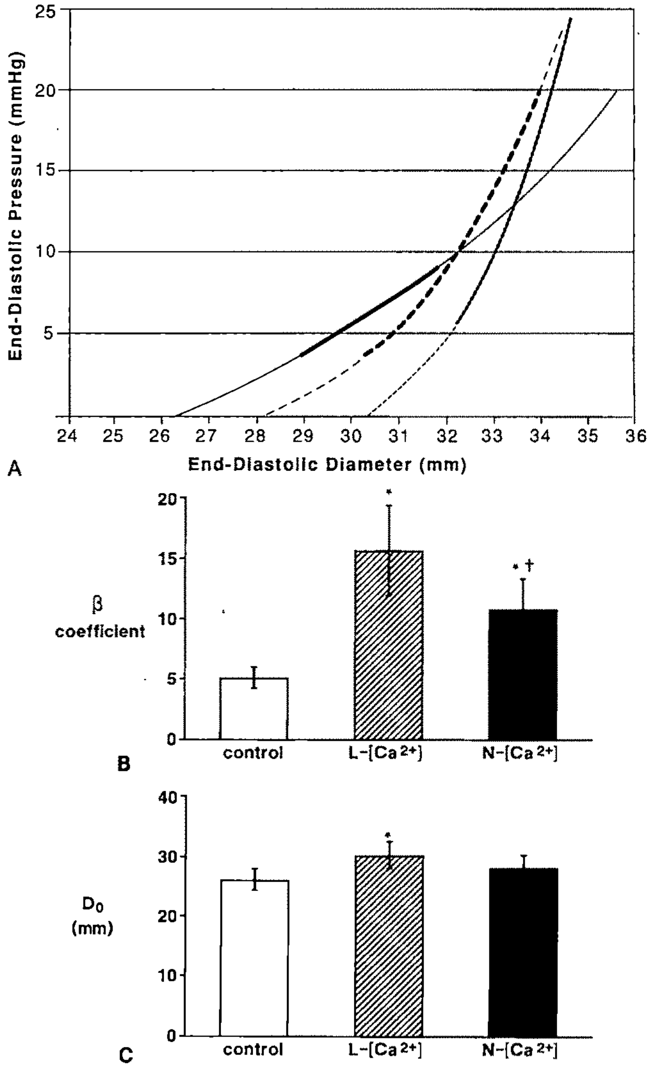

Fig 5. Curves and variables in left ventricular chamber stiffness. (A) Averaged end-diastolic curves of each experimental condition. (solid line = *control;* broken line = *postischemic N-[Ca^{2+}];* dotted line = *postischemic L-[Ca^{2+}].) (B) β Coefficients. (C) Diameter axis intercepts (D_0). Data are shown as the mean ± the standard error of the mean. (* above bar = significantly different from control; † above bar = significantly different from L-[Ca^{2+}] state.)*

ing this critical period of reperfusion could potentiate myocellular injury, thereby resulting in diminished function and metabolism. On the other hand, administration of calcium at this juncture might exert a direct positive inotropic effect leading to improved postischemic ventricular performance and might raise coronary perfusion pressure by vasoconstriction.

In the current study, in which $CaCl_2$ was infused after a period of global ischemia and hypocalcemic reperfusion, the positive inotropic effect predominated, resulting in significantly improved postischemic systolic and diastolic function. In addition, this augmented performance was accomplished without a concomitant increase in oxygen demands because of an observed reduction in ventricular diameter and hence ventricular wall stress, which counterbalanced the oxygen demands of increased contractility. We conclude that administration of $CaCl_2$ to the postischemic myocardium to normalize iatrogenic hypocalcemia or to augment ventricular performance is safe when there has been an intervening period of hypocalcemic reperfusion after cardioplegia.

We chose the afterload reduction technique using right heart bypass for this study for several reasons. First, a large range of data points for P_{es} and SW could be obtained. The LV filling rate was held constant at 80 mL/min/kg to avoid the sigmoid pattern of P_{es}–D_{es} data that was obtained consistently when preload and afterload reductions were used concurrently (unpublished observations). Because the indices used in this study are relatively independent of preload and afterload [8, 9], the profiles of the pressure–diameter relations would be similar to those obtained by other methods. Minor-axis diameter was used [11] instead of calculated or measured volume. In studies [12, 13] in which the LV pressure–dimension relations were compared with simultaneously acquired pressure–volume relations, pressure–dimension relations were shown to respond in a manner similar to pressure–volume relations and to be useful in assessing global changes in contractility.

The functional depression observed in the postischemic hypocalcemic state can be partitioned into two major categories related to calcium kinetics: hypocalcemia-induced ventricular dysfunction and ischemia/reperfusion injury.

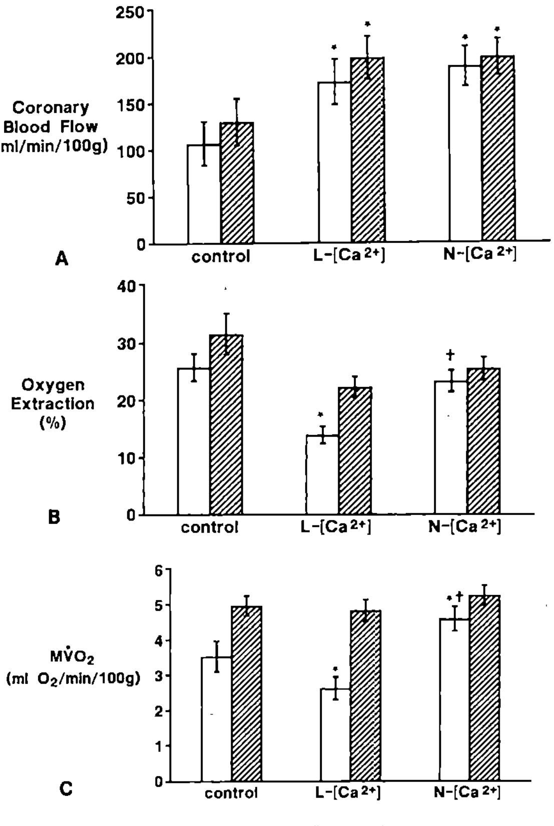

Fig 6. (A) The average coronary blood flow, (B) oxygen extraction, and (C) myocardial oxygen consumption ($M\dot{V}O_2$) *during each experimental condition. Data are shown as the mean ± the standard error of the mean.* (L-[Ca^{2+}] = *postischemic hypocalcemic state;* N-[Ca^{2+}] = *postischemic normocalcemic state;* open bars = *beating empty state;* hatched bars = *beating working state;* * = *significantly different from control;* † = *significantly different from L-[Ca^{2+}] state.)*

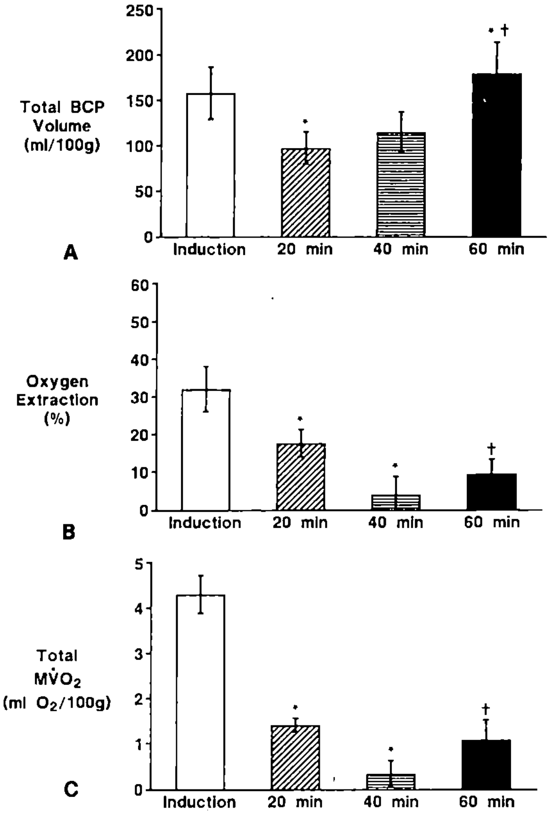

Fig 7. (A) The average total blood cardioplegia (BCP) *volume during each infusion, (B) myocardial oxygen extraction, and (C) myocardial oxygen consumption multiplied by each infusion time* (total $M\dot{V}O_2$). *Data are shown as the mean ± the standard error of the mean. (* = significantly different from induction; † = significantly different from previous value.)*

Role of [Ca^{2+}] Repletion in Hypocalcemia

The partial recovery of cardiac contractility observed with normalization of systemic [Ca^{2+}] suggests that part of this apparent myocardial "dysfunction" is related to the hypocalcemia itself. The magnitude of contractile force is determined in part by the amount of Ca^{2+} in the cytoplasm, which is thought to be derived from a rapidly exchangeable calcium pool [14]. An increase in extracellular [Ca^{2+}] stimulates the excitation-contraction coupling process by serving as a trigger for subsequent calcium release from the sarcoplasmic reticulum [15]. The result of this calcium-related stimulation is enhanced myocardial contraction, the magnitude of which is proportional to the intracellular calcium concentration.

Role of [Ca^{2+}] Repletion in Ischemia/Reperfusion Injury

Postischemic ventricular performance is known to be depressed by several calcium-related mechanisms. Ischemia/reperfusion injury is known to result in a depression of calcium-dependent enzyme activity in membrane systems, including sarcolemma, sarcoplasmic reticulum, and possibly mitochondria. Calcium–adenosine triphosphatase involved in calcium transport in the sarcoplasmic reticulum is depressed after ischemia and reperfusion [16, 17], leading to contractile dysfunction. A recent study by Kusuoka and colleagues [18] demonstrated a depression in calcium sensitivity by the myofibrils as well as a decrease in the inward calcium current during excitation in the ischemic and reperfused myocardium. These postischemic hearts, however, were capable of responding to calcium administration with augmented contractility in a dose-dependent manner [18, 19].

Calcium repletion may enhance contractility in the postischemic heart by stimulating the excitation-contraction coupling process and by increasing the inward calcium current. Both mechanisms lead to a transient elevation of intracellular calcium available for the less sensitive contractile apparatus during systole. In this study, we found that performance of the ischemic and reperfused left ventricle improved with calcium repletion.

Diastolic Properties

Myocardial relaxation, which reflects the rate of calcium removal from the contractile apparatus, essentially depends on the efficiency of the calcium-accumulating activity of the sarcoplasmic reticulum on a beat-to-beat basis [20]. In the ischemic and reperfused heart when ventricular dilation occurs secondary to systolic dysfunction [11], both tissue stiffness and chamber stiffness increase [1, 19, 21], manifested as an increase in end-diastolic pressure and the β stiffness coefficient. However, data from this study indicate that the correction of extracellular hypocalcemia improved not only LV contractility but also LV diastolic distensibility, which is very sensitive to changes in intracellular calcium kinetics. The improved diastolic properties may be due to stimulation of calcium–adenosine triphosphatase localized in the sarcoplasmic reticulum, which is responsible for calcium accumulation. Stimulation of this enzyme would result in greater calcium sequestration and more complete relaxation. This same stimulation of the calcium–adenosine triphosphatase enzymes by the elevation of cytosolic calcium would also augment contractile responses, as already discussed.

In the current study, we found that postischemic [Ca^{2+}] repletion did not increase $M\dot{V}O_2$ in the working heart over and above that in the hypocalcemic state, despite the marked improvement in systolic function. The observed positive inotropic effects of calcium would tend to increase oxygen demands by increasing the rate of contraction and stress development. However, the tendency of these effects to augment postischemic $M\dot{V}O_2$ was apparently counterbalanced, possibly by a decrease in overall wall stress or a decrease in pressure–volume area [13] secondary to a reduction in LV dimensions. This conclusion was confirmed in our study, in which calcium correction decreased both LV systolic ($D_{es,100}$) and diastolic (D_0) dimensions.

The delay in calcium repletion may have played an important role in improving function, rather than being detrimental. The ultimate functional and morphological fate of the ischemic heart is partially determined during the early moments of reperfusion [1, 3] because it is during those moments that myocytes are the most susceptible to injury, including calcium overload. Hypocalcemia during the early phase of reperfusion would avoid calcium-related reperfusion injury. However, a critical point of the calcium correction strategy is its timing [6, 19, 22]. Because the magnitude of calcium accumulation is dependent on the severity of the preceding ischemic insult and can also be reduced by a modification of the reperfusion method, the appropriate time to replenish calcium can vary between each instance. Shen and Jennings [3] demonstrated that, in canine hearts subjected to 40 minutes of regional ischemia followed by reflow, tissue calcium uptake increased linearly with time and peaked at ten minutes after reperfusion. Numerous studies also suggest that the various manifestations of reperfusion injury are expressed after ten to 30 minutes of reflow. Although we waited a total of 60 minutes to normalize calcium levels, this period is not obligatory and can be reduced considerably to expedite surgical progress.

In summary, we found that [Ca^{2+}] correction in the postischemic reperfused heart restored LV performance without additional metabolic cost. Clinical trials of premature versus staged inotropic support in general—and [Ca^{2+}] correction in particular—are warranted. However, the effects of calcium administration to hearts with acute evolving infarction necessitating coronary artery bypass grafting have to be clarified. In this setting, calcium administration carries the potential for increasing *local* metabolic requirements secondary to inotropic recruitment of contractile reserve, and hence the potential for enlarging the infarct. Whether the delay in calcium administration will circumvent these potential hazards also must be determined. However, we have demonstrated that $CaCl_2$ can be administered after global ischemia without precipitating functional deterioration or adversely affecting the oxygen supply–demand balance.

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DISCUSSION

DR ROBERT A. GUYTON (Atlanta, GA): The one question I have concerns the fact that you waited one hour after reperfusion before bringing the calcium levels back up to normal. I think there are data from Duke indicating that administration of calcium at the time of reperfusion leads to elevated myocardial calcium levels, but that waiting for ten minutes does not. I know that most of us cannot wait for an hour after reperfusion before bringing calcium levels back up. Do you think ten minutes is long enough?

DR YOKOYAMA: The outcome of reperfusion injury is almost determined within ten minutes under artificial reperfusion, so we can safely add calcium about ten minutes after the onset of reperfusion. However, to avoid the unnecessarily enhanced mechanical work, it is best to wait for at least 20 to 30 minutes to direct the supplied oxygen to the cellular reparative process.

PRESIDENT RICHARD E. CLARK: Because magnesium is a bivalent ion also, have you looked at magnesium levels in the same way you did calcium levels?

DR YOKOYAMA: Magnesium has a so-called calcium-antagonist effect, and it decreases the calcium entry. In this experiment, we did not add magnesium to the BCP, and it was maintained in a lower concentration than normal, so that we examined only the effect of calcium.

DR GEORGE R. DAICOFF (St Petersburg, FL): As it takes a while to obtain your data, the volume–pressure loops and the other studies, how do you know that it is not the time factor that is allowing the hearts to get better? For example, have you done the low calcium study twice in a row?

DR YOKOYAMA: Are you asking about the effect of time on contractility?

DR DAICOFF: It looks as though you did your studies with low calcium, and then allowed a period when the hearts are still being perfused and are still recovering. How do you know that that time itself is not important in restoring left ventricular function?

DR YOKOYAMA: After data collection during the hypocalcemic state, we added $CaCl_2$ and waited five or ten minutes to allow stabilization of normocalcemia before the next data collection. I think the time is short enough to negate any time effect on recovery of myocardial function.

Reoperation in the Intensive Care Unit

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From July 1, 1984, through June 30, 1989, after 1,259 open heart operations, 110 patients (8.7%) underwent 162 early reoperations either in the intensive care unit (144 procedures) or in the operating room (26 procedures). Reexploration for bleeding (49 procedures) (3.9%) and intraaortic balloon removal (50 procedures) (4.0%) were the two most common procedures. Ninety percent and 96% of these procedures, respectively, were performed in the intensive care unit. Mediastinal infections occurred in 4 (6.1%) of 66 patients undergoing repeat mediastinal operations for all indications. No infection occurred after reexploration for bleeding nor did mediastinal infection occur after reoperation in the intensive care unit. Postoperative death in these 110 patients was not related to reoperation except possibly in the case of 1 patient (0.9%). Average transit time to and from the operating room for patients returned there for reoperation was 89.7 minutes. Charges for procedures performed in the operating room were at least twice as great as for those performed in the intensive care unit. This experience supports expanded use of reoperation in the intensive care unit, as it is safe, effective, economical, and convenient.

(Ann Thorac Surg 1990;49:903–8)

We have had experience with reoperation in the intensive care unit (ICU) for more than 25 years. Chiefly, this has dealt with resuscitation or reexploration for bleeding. Initially, these operations were emergency procedures, which precluded return to the operating room (OR). Encouraged by our early results and after initiation of a cardiac surgical service at an affiliated institution, we gradually expanded the use of reoperation in the ICU. This report is an analysis of our experience on that affiliated service.

Material and Methods

Patient Population

From July 1, 1984, through June 30, 1989, 1,259 patients underwent open heart operations on our affiliated service. One hundred ten (8.7%) of these patients had 162 reoperations. Over the 5-year period, the annual incidence of reoperation ranged from 4.3% to 13.4%. One reoperation was required in 73 patients (66.4%), two reoperations in 29 patients (26.4%), and three or more in 8 patients (7.3%). The majority of patients had their operations performed in the ICU, but 24 similar operations were performed in the OR by choice of the surgeon. Some patients had similar operations performed in both places. Some had more than one procedure performed at the same operation. Patients whose first operation was performed in either the ICU or the OR were listed in that location for purposes of analysis of the patient profile.

Operations usually performed in the OR, such as exploratory laparotomy, gastrectomy, feeding enterostomy, closure of a pulmonary air leak, construction of an arteriovenous fistula, and permanent transvenous pacemaker insertion, were excluded from this analysis. Patients in whom a lateral thoracotomy was used for the initial operation were returned to the OR if reoperation through this incision was required. Percutaneous insertion and nonoperative removal of an intraaortic balloon (IAB) were also excluded. Extracorporeal membrane oxygenation support, established in conjunction with ICU resuscitation, was not considered a separate procedure.

The time for each operation was tabulated. Hospital charges for the OR or ICU for each procedure performed were computed for each operation done at that location. The included charges were for room, equipment, and personnel. Because the amount of supplies used was thought to be similar for each location, these charges were not included. Professional charges were not included for similar reasons. The charges for each procedure were totaled, averaged, and compared. Because transportation from the ICU to the OR and back is required when the procedure is performed in the OR, the times for each of these operations were recorded, tabulated, and averaged.

Data were analyzed using the Statview 512$^+$ statistical software package (Brainpower, Inc, Calabasas, CA). The χ^2 test and Fisher's exact test were used to determine significance for discrete variables. Continuous variables were analyzed using Student's t test. A p value of less than 0.05 was considered significant.

Technique

The technique for reoperation in the ICU is simple. General OR routine is followed except that the patient remains in the ICU bed with all monitoring equipment attached. The patient and bed are cleared of any extraneous wires, lines, or equipment. If the median sternotomy is to be reopened, the electrocardiographic electrodes are

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changed from the chest to a more remote location. The arms of the patient are usually brought alongside the head to allow access to both arterial and venous lines in either arm. The hub of the pulmonary artery catheter is also placed at the head of the bed. The temporary pacing electrodes, placed at the previous operation, are attached to a pacemaker, which should be easily accessible.

An anesthesiologist or nurse, stationed at the head of the bed, can easily administer appropriate medications and fluids in addition to monitoring the patient. We have found a combination of diazepam, morphine sulfate, and pancuronium bromide a most useful anesthetic combination for these procedures. Medications are administered by bolus injections through the pulmonary artery line for more rapid distribution.

Ventilation is continued with a volume-cycled respirator at the established settings, except that the fractional concentration of oxygen in inspired gas is increased to 100% and positive end-expiratory pressure is maintained at 5.0 cm H_2O or lower. A cautery grounding pad is attached appropriately. Coagulation defects are corrected by administration of appropriate blood products before, during, and after reexploration, if necessary. Systemic cephalosporin coverage is continued perioperatively.

The technique of reoperation performed in the ICU is identical to that in the OR. All operating personnel wear surgical caps, masks, gowns, and gloves. All other associated personnel wear caps and masks. After the area has been prepared with a povidone-iodine solution, the operative field is draped, and the chest is reopened. The soft tissues and sternal edges are closely inspected for bleeding points; liberal use is made of the electrocautery for their control. If either pleural space has been opened, that space is evacuated of all blood and fluid. Any clot within the pericardium is evacuated, and the pericardium and pleural space are irrigated with warm saline solution.

Systematic inspection of all operative sites is then undertaken. Suture lines, anastomoses, conduits, and cannulation sites are each visualized in turn. Each vein and internal mammary artery graft is examined to detect any bleeding from side branches. The heart is mobilized from the pericardium to inspect each distal anastomosis. Appropriate retraction of the sternal halves allows satisfactory visualization of these areas without rotation of the patient or the bed. A fiberoptic headlight and optical magnification are essential for accurate visualization. Bleeding sites are controlled by sutures, ligatures, stainless steel clips, cautery, or application of thrombostatic material as required. The drainage tubes are cleared of any clotted blood. After hemostasis has been achieved, the wound is irrigated with a dilute antibiotic solution and closed in the routine manner. Modifications may be necessary depending on the incision to be reopened and the speed with which it is necessary to proceed.

The ICU is stocked with sutures, sponges, drapes, gowns, and a standard cardiovascular instrument pack. A standard electrocautery and adequate suction are provided. Occasionally, equipment for autotransfusion has been used. Intensive care unit nursing personnel, trained in basic OR technique, assemble appropriate equipment and administer various necessary agents including blood. Trained cardiovascular OR nurses assist in performance of the procedure. Both groups of nurses are essential to the success of this technique.

Table 1. Profiles of the Two Patient Groups[a,b]

Variable	Intensive Care Unit[c] (n = 95)	Operating Room[c] (n = 15)
Age (yr)		
Mean ± SD	63.9 ± 9.4	66.0 ± 10.0
Range	37–81	48–81
Sex		
Male	59 (62.1)	8 (53.3)
Female	36 (37.9)	7 (46.7)
Prior operation		
CABG	72 (75.8)	11 (73.3)
AVR	11 (11.6)	3 (20.0)
MVR	10 (10.5)	1 (6.7)
Other	2 (2.1)	0 (0)

[a] There were no significant differences between the groups. [b] Numbers in parentheses are percentages. [c] This was the site of the first reoperation.

AVR = aortic valve replacement; CABG = coronary artery bypass grafting; MVR = mitral valve replacement; SD = standard deviation.

Results

There was no significant difference in age, sex, or types of prior operation between patients undergoing reoperation in the ICU and those having reoperation in the OR (Table 1). The types of procedures performed are listed in Table 2. Femoral artery reconstruction or thrombectomy at the time of IAB removal was the usual reason multiple procedures were performed at the same operation. One additional patient had femorofemoral bypass grafting in the ICU to relieve leg ischemia secondary to IAB insertion. One of the 2 patients returned to the OR for removal of an IAB had had it inserted at the original operation through the ascending aorta.

Table 2. Procedures Performed at 162 Reoperations

Procedure	ICU	OR	Total
Mediastinal reexploration (bleeding)	44	5	49
IAB removal	48	2	50
Tracheostomy	21	5	26
Sternal rewiring	6	11	17
Resuscitation	11	0	11
Femoral artery reconstruction or thrombectomy	10	2	12
Other	4	1	5
Total	144	26	170

IAB = intraaortic balloon; ICU = intensive care unit; OR = operating room.

Reoperative Procedures

MEDIASTINAL REEXPLORATION. Mediastinal reexploration for bleeding or cardiac tamponade was performed 49 times in 39 patients. One reexploration was required in 31 patients, and 6 patients required two and 2 patients required three reexplorations. Of the 8 patients requiring multiple reexplorations, 50% had had valve replacement and the others, coronary artery bypass grafting. Of patients requiring single reexploration for bleeding, 79.5% had had prior coronary artery bypass grafting. All but one of the multiple reexplorations were performed in the ICU.

The indication for reoperation was excessive chest tube drainage in 13 instances (26.5%). All of these patients were in hemodynamically stable condition without evidence of cardiac tamponade or hypotension. Cardiovascular instability was the indication in 28 instances (57.1%) and cardiac tamponade in 31 (63.3%); both were present on 23 occasions (46.9%). Four of the 5 patients returned to the OR were in hemodynamically stable condition. Three of these 5 had hemodynamic measurements consistent with early cardiac tamponade. Two were in hemodynamically stable condition, and 1 was not.

The time until reoperation for the group reoperated on in the ICU ranged from one hour to 32 hours (mean time, 8.0 hours; median time, 7.5 hours). For those returned to the OR, the time ranged from three to nine hours (mean time, 5.6 hours; median time, six hours). In the ICU group, the chest tube drainage during that interval ranged from 60 to 2,233 mL/h (mean drainage, 459 mL/h; median drainage, 355 mL/h). Comparable volumes for patients returned to the OR ranged from 225 to 1,158 mL/h (mean drainage, 588 mL/h; median drainage, 496 mL/h). Shed blood was salvaged and reinfused whenever possible. The estimated blood volume evacuated at ICU reexploration ranged from 0 to 4,000 mL (mean volume, 926 mL; median volume, 600 mL). In those returned to the OR, volumes ranged from 100 to 2,000 mL (mean volume, 1,020 mL; median volume, 1,000 mL).

No specific bleeding source was found on 27 occasions (55.1%). Two bleeding sites were identified in five instances. Graft side branch or anastomosis was the source identified 12 times, and bleeding originated from the chest wall on six occasions, the pericardium twice, and the diaphragm once. In six other instances, various other sources were thought to contribute to the continued bleeding on one occasion each. Cannulation sites were never found to be a source of bleeding. There were no differences in bleeding site incidence between patients reoperated on in the ICU and those seen in the OR; therefore, the patients were combined for this analysis. On 38 occasions, coagulopathy related to medication (aspirin, warfarin sodium, heparin sodium, dipyridamole, nonsteroid, antiinflammatory agents, or thrombolytic agents) was believed to contribute to, if not to be entirely responsible for, the bleeding that was encountered. These coagulation abnormalities were corrected by administration of various blood components.

Sutures, ligatures, or stainless steel clips were used to control the bleeding sites on 22 occasions. Thrombostatic oxidized cellulose was employed on nine occasions, and application of topical thrombin or fibrin glue was used eight times. The electrocautery was used liberally.

Ten (25.6%) of the 39 patients reexplored for bleeding died in the postoperative period. Eight died of cardiac-related causes, either arrest or failure, and 1 each, of a cerebrovascular accident and respiratory insufficiency from one to 85 days postoperatively (mean time, 15.1 days; median time, five days). All 10 had been reoperated on in the ICU. There were no deaths due to uncontrolled surgical bleeding. There were no mediastinal infections after reexploration for bleeding. Multiple reexplorations for bleeding did not increase mortality (25.0%, multiple, and 25.8%, single). One survivor of two reexplorations for continued postoperative bleeding sustained rupture of the posterior left ventricle after mitral valve replacement. Repair was accomplished in the ICU.

INTRAAORTIC BALLOON REMOVAL. Removal of an IAB was performed as frequently as mediastinal reexploration for bleeding. The IAB is inserted intraoperatively into an exposed femoral artery using the percutaneous technique. Therefore, it is removed later at operation with repair of the femoral artery insertion site. Femoral artery thrombectomy or reconstruction with an onlay patch or a combination of these has been associated almost exclusively with IAB removal whether performed in the ICU or the OR.

RESUSCITATION. Eleven patients had resuscitation with reopening of the median sternotomy. All underwent operation initially in the ICU. Four patients were supported by extracorporeal membrane oxygenation, cardiac assist devices, or both. All 4 patients eventually died. In 7 of the 11 patients, an IAB was inserted either at the initial operation or during resuscitation in the ICU. One of them survived. The overall survival in this group was 18% (2 patients). All deaths were due to cardiac-related causes. There were no mediastinal infections either in survivors or in nonsurvivors.

STERNAL REWIRING. The sternum was rewired in 16 patients. There were two deaths (12.5%) in this group. One patient died of respiratory and renal failure 37 days after rewiring and the other, of cerebrovascular injury and multisystem organ failure 22 days after rewiring.

Sternal dehiscence, with or without minor skin separation, occurred in 7 patients and was repaired in the ICU on five occasions and in the OR twice. The only postoperative infection developed in a patient in whom there had been some skin separation and in whom rewiring had been performed in the OR. All 7 patients survived with healing of the median sternotomy.

Six patients had secondary sternal closure because hemodynamic compromise prevented sternal reapproximation at the original operation. If the skin could not be closed completely, the remaining skin defect was closed temporarily with a patch fashioned from an Esmarch tourniquet. In 1 of these patients, secondary sternal reapproximation had been unsuccessful because of persistent hemodynamic compromise. Sternal closure was accomplished successfully several days later. Although

Table 3. Average Charges for Procedures

Procedure	Charges
Mediastinal reexploration (bleeding)	
OR (5)	$894
ICU (44)	$413
Sternal rewiring	
OR (11)	$910
ICU (6)	$407
IAB removal	
OR (2)	$916
ICU (48)	$202
Tracheostomy	
OR (5)	$812
ICU (21)	$112

IAB = intraaortic balloon; ICU = intensive care unit; OR = operating room.

there were no postoperative mediastinal infections in these 6 patients, 1 patient died of respiratory and renal insufficiency on postoperative day 37. Three of these procedures were done in the OR and the remainder in the ICU.

Three patients with infected sternal dehiscence underwent debridement and reclosure in the OR. Although all wounds healed primarily, 1 patient died of cerebrovascular injury and multisystem organ failure 22 days postoperatively.

TRACHEOSTOMY. Tracheostomy was performed on 26 occasions, 21 times in the ICU and five times in the OR. There were three late deaths in this group; 1 patient each died of multisystem organ failure, respiratory insufficiency, or renal failure and sepsis.

Hospital Charges

Average hospital charges for each procedure were calculated. The charges for reexploration, sternal rewiring, IAB removal, and tracheostomy are tabulated in Table 3. Charges occurring in the OR are substantially higher than those in the ICU. Removal of an IAB was performed in the OR only twice. One of these patients had removal of an IAB that had been inserted through the ascending aorta. Because of this and the small number of operative procedures performed in the OR in this group, comparison of charges should not be made. However, the charge for IAB removal in the ICU is low. Charges for other procedures reflected similar differences but are not shown because the smaller numbers of procedures performed do not allow reasonable comparison.

Transportation Time

The time for transportation to and from the OR was recorded and tabulated for each of 24 operations. The average time required for transportation to the OR from the ICU was 45.3 minutes. This does not include the time required to prepare the monitoring lines, electrocardiographic leads, and oxygen delivery lines prior to leaving the ICU. The average time for transportation back to the ICU from the OR after operation was 44.4 minutes. This time does not include reestablishment of the hemodynamic monitoring lines and electrocardiographic leads once the patient was returned to the ICU. The mean total transportation time was 89.7 minutes.

Comment

In 1984 we initiated a cardiac surgical service at one of our affiliated institutions. It was organized somewhat differently from our service at the university hospital and resembled more closely a private service in a community hospital. We therefore modified several aspects of patient management to accommodate the organization and experience at that institution. One area that was reevaluated and expanded was the role of reoperation in the ICU. As our experience increased, we found the technique to be effective and convenient without increasing morbidity or mortality when applied for specific indications.

Reexploration in the ICU for resuscitation has been performed for many years. Our experience with this indication has not been dissimilar to that reported by others [1, 2]. Patients with sudden cardiac arrest, usually due to arrhythmia, are frequently successfully resuscitated [2]. We, as well as others [1, 2], have shown that patients with cardiac arrest due to cardiac muscle failure have a lower survival rate, as the arrest is frequently the terminal event. Often these patients are explored with the hope of finding a remediable situation. Since the development of cardiac assist devices and extracorporeal membrane oxygenation, they have been employed in these resuscitative efforts. Although their use has been encouraging, improved patient salvage has not been observed in all circumstances [3].

We [4–6] have documented that cardiac procedures currently are being performed more frequently in patients at higher risk. However, under these circumstances, reexploration, which can include use of a cardiac assist device, remains the only acceptable alternative in these desperately ill patients [3].

Mediastinal reexploration for bleeding usually is undertaken when excessive postoperative blood loss occurs or there is evidence or suspicion of cardiac tamponade. In our early experience, such patients were routinely returned to the OR when possible. However, as experience increased, we began to reexplore more patients in the ICU on a less-urgent basis. Currently, this is done almost routinely. Our experience has demonstrated this approach to be effective, less costly, and convenient without increasing morbidity and mortality. Infections have not occurred in these patients after reexploration. McKowen and associates [2] commented on the low incidence of infection under these circumstances.

The cause of death in patients undergoing reoperation in the ICU was primarily cardiac related. However, the one death that might be attributed to reoperation in the ICU occurred in this group. A 63-year-old man was reexplored for bleeding after coronary artery bypass grafting and became severely hypertensive during the procedure. He evidenced a brainstem infarction after reexploration. This 1 case emphasizes the importance of avoiding hypertension in these patients by appropriate use of anesthesia and vasodilators. These critically ill patients have tolerated mediastinal reexploration exceptionally well without the cumbersome and inconvenient maneuvers associated with a return to the OR. Monitoring during transportation usually is less extensive than in either the OR or ICU. Hemodynamic monitoring is uninterrupted when patients remain in the ICU. Although we have found no discernible disadvantage to reoperation in the ICU, the lack of interruption in hemodynamic monitoring has proved to be one of the major advantages of this technique in critically ill patients.

We do not believe that every reoperation should be performed in the ICU. Although a patient with a median sternotomy is readily suited for ICU reexploration, it is quite difficult to effectively reexplore a patient through a lateral thoracotomy in the ICU. A fiberoptic headlight is essential to the success of this technique. When a patient is reexplored through a prior median sternotomy for mediastinal bleeding, it usually is not necessary to rotate the patient from side to side. Indeed, some of the patients returned to the OR have been reexplored in their bed rather than on an operating table. This satisfactory experience was another factor that encouraged us to reexamine indications for reoperation in the ICU. If a situation occurs that cannot be handled in the ICU, the patient should be transported to the OR so that the problem can be dealt with adequately. Although this was never necessary in the experience reported here, we would not hesitate to return a patient to the OR if it were considered necessary.

Our group [7] has previously reported our unfavorable experience with percutaneous IAB insertion in situations in which there is a poorly palpable or impalpable arterial pulse. This has occurred chiefly during operation, particularly during attempted weaning from extracorporeal circulation. In these circumstances, we insert the IAB using the percutaneous technique under direct vision in an exposed femoral artery and have found this method to be quite satisfactory. When the IAB has been inserted in this manner, we have removed it at operation and repaired the arterial insertion site. Depending on the operative findings at the time of removal, thrombectomy or arterial reconstruction with an onlay patch or a combination of these may be necessary and usually is easily accomplished in the ICU. Removal of an IAB inserted in this manner has been one of the more frequent indications for ICU reoperation. The operation is brief, effective, and economical. On occasion, when the femoral artery was observed to be severely diseased at IAB insertion, the patient was returned to the OR for removal.

We have found tracheostomy can be performed easily, rapidly, and less expensively in the cardiac surgical ICU. Other less frequently performed procedures such as femorofemoral bypass also have been accomplished easily in this setting without a procedure-related increase in morbidity or mortality.

Hospital charges for procedures performed in the OR are greater than those for procedures performed in the ICU. For mediastinal reexploration, these cost differences were of the same order of magnitude reported by McKowen and associates [2]. They reported costs; we reported charges. Some of the differences between the reported figures of McKowen and colleagues and ourselves could be due to a difference between cost and charges. Another reason for the difference could be our exclusion of supply charges. Because we considered them to be the same for each location and as we were interested mainly in charge differences between locations, supply charges were excluded in our analysis. Their inclusion would increase the total charge, however. Depending on the procedure analyzed, the reduction in charges to our patients ranged from $500 to $700 per procedure compared with the $1,100 cost reduction reported by McKowen and co-workers [2]. A reduction in charges of $500 per procedure for 162 operations is a total savings of $81,000, and a $700 reduction per procedure would be $113,400.

Although economy in health care delivery currently is receiving a good deal of attention, cost savings should not be the foremost reason for modifying surgical practice. Neither should convenience be a primary consideration. Effectiveness of treatment and patient safety are the major factors to be weighed first. Only after these two requirements are satisfied should economy and convenience be considered. Our premise is not to supplant the OR as the location for performing operations but rather to suggest that ICU reoperation is an acceptable alternative in appropriately selected patients. Our experience indicates that the ICU can be regarded as a suitable alternative site for reoperation because morbidity and mortality are not increased. Furthermore, as we also have demonstrated its effectiveness, economy, and convenience, we believe expanded use of the ICU as a site for reoperation should be encouraged.

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DISCUSSION

DR HARRY SIDERYS (Indianapolis, IN): I had the opportunity to review the manuscript and found that our experience very closely parallels that at your hospital. My co-workers and I became aware that we had operated in the ICU as long as 15 years ago when we had patients in acute tamponade who could not tolerate the trip to the OR. Over the years, we have gradually improved our facilities with headlights and all the various equipment necessary to do a good operation in the ICU. Our experience is very similar to yours in the number of procedures and the kinds of procedures done there.

I would like to mention one procedure that we have been doing for the last 4 years that was not listed in your study and has been very helpful to us. In patients who have severe aortoiliac disease and require placement of an IAB or patients who must have removal of a balloon in a femoral artery because of vascular compromise, we place the balloon through the axillary artery in the ICU through a cutdown. The radiologist helps us, as does the C-arm fluoroscopic unit. We guide first the wire and then the balloon into the descending thoracic aorta and are able to free up the groins and obtain good balloon inflation. We have also performed this in the operating theater. In that case, with the sternum open, we guide the balloon down the left subclavian artery into the arch manually. These patients do not have to be taken back to the OR for balloon removal. That can be done very simply in the ICU setting.

All these ICU procedures have helped us take care of patients, and I agree with you that we ought to explore them further.

DR KIT V. AROM (Minneapolis, MN): I enjoyed the paper very much and would like to take this idea home and start doing the same thing. I have a couple of questions. First, for a real emergency—the one that has to be done in the middle of the night—do you have OR personnel go to the ICU, or do you use your own ICU nurse? Second, have you done all these procedures at the bedside, or do you have in the ICU a small room set aside that looks like a small OR?

DR NORMAN J. SNOW (Cleveland, OH): I have a couple of comments. First, our experience differs from this. I do not remember any instance when we took 80 minutes to transfer a patient to and from the OR, which happens to be adjacent to our ICU. I think that a lot of this is facility dependent. We would never have been able to do what you do in our old ICU, though we could easily operate in our new one. It is very important to individualize this to the facility available.

Second, could you comment on whether or not the safety of the outcome of these procedures is overwhelmed by the patient's condition rather than the site of the operation? If the patient is very sick, he or she is liable to die no matter where you do the procedure. If the patient is not that sick and there is bleeding of 300 mL/h that you can autotransfuse, the patient will live no matter where you do the procedure.

DR KAISER: I will answer the questions in reverse order, as that would be easier.

Dr Snow, you are correct. I tried to point out in the presentation that the mortality with this technique is not procedure related. It is related to the associated conditions, those underlying conditions for which the patient received the initial operation and also those after the initial operation. We believe that use of this technique should be individualized.

As for the time required to transfer patients, there is a lot of scurrying around when this is done. I also was surprised to learn that it took so long to move patients back and forth. However, there is a lot to be done when changing wires and monitoring devices and transporting the patient, and all this does take time. You are again correct that sometimes it may be a long distance from the ICU to the OR. At both institutions where I work we have to use an elevator. Traversing that distance does consume time, but it is a relatively small proportion.

Dr Arom, your comments and questions about the technique are pertinent. We do operate on the patient in bed. This has the disadvantage of not being able to roll the patient, but for a median sternotomy, we can usually elevate one side of the sternum or the other to see any potential bleeding point in that area. For most of the other procedures, it is not necessary to rotate the patient. We do use a headlight. In one of our institutions, not included in this report, we have small OR lights in the ceiling of each ICU room. We do not use a special room; reoperation is performed in the patient's room in the ICU.

In the institution from which this report is generated, we have cardiothoracic surgical nurses on call. They are notified as early as possible, and their response is quite prompt. However, we can begin reoperation on someone who needs reexploration very quickly with the help of well-trained ICU nurses, who assemble the packs, prepare the patient, and so on. By the time some additional assistance is required, the OR people are there. We have found this arrangement to be very effective. It is important to emphasize that well-trained, cooperative, and enthusiastic nurses are essential to the success of this technique.

Dr Siderys, thank you for your comments. My associates and I have learned many operative pearls from you. We have used your axillary artery technique for IAB insertion. One of my associates, Dr McBride, has modified this to provide ambulatory IAB pumping in cardiac transplantation candidates on the waiting list.

The major point of this presentation is that these procedures can be safely and effectively performed in the ICU. The cost benefit is secondary, although welcome.

Predicted Pulmonary Function and Survival After Pneumonectomy for Primary Lung Carcinoma

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Between 1982 and 1987, 139 patients with primary carcinoma of the lung were treated with pneumonectomy. Thirty-nine patients (28%) were in clinical stage I, 10 (7%) were in clinical stage II, and 90 (65%) were in clinical stage III. Overall actuarial 3-year survival was 33%. Actuarial 3-year survival for patients in clinical stage I was 44%; for those in clinical stage II, 48%; and for those in clinical stage III, 28%. Risk factors for operative mortality examined included preoperative forced vital capacity (FVC) of 2.13 L or less and forced expiratory volume in 1 second (FEV_1) of 1.65 L or less, percent predicted FVC of 64% or less and FEV_1 of 65% or less, predicted postoperative FVC of 1.31 L or less and FEV_1 of 0.89 L or less, and predicted postoperative percent predicted FVC of 41% or less and FEV_1 of 34% or less. Operative deaths occurred only in clinical stage III patients (7/90 or 8%). Patients with compromised pulmonary function based on one or more of the examined risk factors were at increased risk for death (2/10) compared with patients with better pulmonary function (5/80 or 6.25%). Actuarial 3-year survival for high-risk clinical stage III patients ranged from 0% to 16% compared with 28% for other clinical stage III patients. Thirty-day mortality for pathological stage III patients was 6.3% (5/79), and 3-year actuarial survival was 24%. No patient in pathological stage III who was at high risk survived beyond 3.1 years. Select individuals with adequate pulmonary function and stage III disease can achieve substantial long-term survival after pneumonectomy. Patients should not be excluded from pneumonectomy based on stage alone or on the results of any single pulmonary function study.

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After the successful one-stage pneumonectomy for carcinoma of the lung reported by Graham and Singer [1] in 1933, pneumonectomy became the standard treatment of primary carcinoma of the lung for the next two decades. In 1950, Churchill and associates [2] presented improved results with lobectomy, which remains the procedure of choice in the majority of patients with carcinoma of the lung. Surgical resection including pneumonectomy continues as the key to potentially curative therapy for patients with primary lung carcinoma.

Selection of patients who will optimally benefit from resection can be difficult. In patients with compromised cardiopulmonary function, pneumonectomy is associated with substantial risk and mortality [3–5]. In past studies, operative mortality after pneumonectomy ranged from 6.2% to 12.4% [3, 6–8] and 5-year survival, from 8% to 25% [3, 7, 9] for all stages of bronchogenic carcinoma. Historically, patients with stage III bronchogenic carcinoma treated by pneumonectomy achieve a 5-year survival rate of 10% to 13.8% [9, 10].

Recently, our group [11] identified specific criteria associated with perioperative morbidity and mortality after pneumonectomy for several types of cancer. In this current study, stage-specific survival was analyzed for all patients with primary bronchogenic carcinoma who underwent pneumonectomy for cure to determine if patients with clinical or pathological stage III primary lung cancer benefit from surgical resection and whether preoperative pulmonary function studies can predict postoperative mortality and long-term survival.

Material and Methods

Between 1982 and 1987, 139 patients underwent pneumonectomy for primary carcinoma of the lung at The University of Texas MD Anderson Cancer Center. There were 99 men with an average age of 56.9 years (range, 38 to 80 years) and 40 women with an average age of 58.0 years (range, 39 to 72 years). All patients were evaluated by physicians from the Departments of Thoracic Surgery and Thoracic Medicine after referral by the patient's primary physician.

Clinical staging by the TNM classification [12] was performed by extensive preoperative evaluation and included plain chest roentgenograms and computed tomographic scans of the chest and abdomen. Mediastinal lymph nodes greater than 1 cm in any diameter were categorized as enlarged and defined as clinical N2 disease. Bone scans and computed tomographic scans of the brain were done if symptoms were present. The patient underwent pulmonary function tests including regional pulmonary ventilation and perfusion using xenon 133 gas. All pulmonary function tests were performed by the same laboratory for internal consistency. Details of the xenon

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133 pulmonary function test have been previously described [13–15].

Operative Technique

The staff surgeon was the final arbiter of operative risk and potential resectability before surgical intervention. Central venous pressure, radial artery pressure, pulse oximetry, and urine output were monitored continuously throughout the operation. The operative technique consisted of a serratus anterior muscle–sparing posterolateral thoracotomy, and the extent of resection was determined by tumor size and local extension.

Pneumonectomy was performed in all patients whose tumor was considered potentially completely resectable. Mediastinoscopy was not routinely performed. The necessity for pneumonectomy was determined at the time of operation by the surgeon. All patients underwent a mediastinal lymph node dissection. Removal of all identifiable nodes along the pulmonary ligament and in the subcarinal regions, tracheobronchial angle, and paratracheal regions was performed. Hilar and interlobar nodes were sampled during anatomical dissection [12], and additional lymph nodes were removed after removal of the lung.

Survival Analysis and Risk Factors

Actuarial survival analysis was performed by clinical stage, pathological stage, and risk factors. Risk factors examined for operative mortality after pneumonectomy included preoperative forced vital capacity (FVC) of 2.13 L or less, preoperative forced expiratory volume in 1 second (FEV_1) of 1.65 L or less, percent predicted FVC of 64% or less, and percent predicted FEV_1 of 65% or less. Using the percent ventilation to the noninvolved lung from the xenon 133 ventilation/perfusion study, predicted postoperative pulmonary function was determined. Postoperative risk factors examined included predicted postoperative FVC of 1.31 L or less, predicted postoperative FEV_1 of 0.89 L or less, predicted postoperative percent predicted FVC of 41% or less, and predicted postoperative percent predicted FEV_1 of 34% or less. No patient was excluded from operation based on any individual test result.

Operative death was defined as death within 30 days of pneumonectomy or death during hospitalization after pneumonectomy at any time. Any patient in one or the other category was counted as an operative death.

Follow-up

All patients were followed until death. Median follow-up is 4.1 years for surviving patients.

Statistical Analysis

Patient survival distributions were calculated by the method of Kaplan and Meier [16]. Differences in survival were calculated using the log-rank test. Differences in operative mortality based on pulmonary function were calculated using the χ^2 tests. All p values are two tailed.

Results

Sixty-five patients (47%) had squamous cell carcinoma; 50 patients (36%), adenocarcinoma; 9 patients (6.5%), adenosquamous carcinoma; 5 patients (4%), large cell carcinoma; and 2 patients (1%), bronchoalveolar carcinoma. Eight patients (6%) had unclassified primary carcinoma of the lung. Seventy-two patients underwent a right pneumonectomy (52%) and 67 (48%), a left pneumonectomy.

Mortality and Morbidity

The 30-day mortality after pneumonectomy was 5% (7 patients). All seven deaths occurred among patients who had a right pneumonectomy (operative mortality, 9.7% [7/72]). There were no operative deaths among patients in clinical stage I or II. Operative mortality for clinical stage III patients was 7.8% (7/90) (Table 1) and was precipitated by an acute cardiac-related event in 4 patients (57%) with death eventually resulting from pneumonia. Two additional patients (29%) died of pneumonia and 1 patient (14%), of a bronchopleural fistula.

Surgical complications included arrhythmias (21 patients, 15.1%), cardiac failure (14 patients, 10.1%), pneumonia (9 patients, 6.5%), hemorrhage (6 patients, 4.3%), pericarditis (5 patients, 3.6%), minor atelectasis (5 patients, 3.6%), and wound infection (2 patients, 1.4%).

Survival and Clinical Stage

Actuarial survival was determined for all patients (Fig 1) and by clinical stage (Fig 2). Overall survival was 23% at 5 years. Thirty-nine patients (28%) were in clinical stage I, 10 (7%) were in clinical stage II, and 90 (65%) were in clinical stage III. Patients with clinical stage I disease had a 44% 3-year survival (median survival, 2.66 years). Patients with clinical stage II disease had a 48% 3-year survival (median survival, 1.97 years). Patients with clinical stage III disease had a 28% 3-year survival (median survival, 1.27 years).

Survival and Pathological Stage

After operation, patients were restaged based on pathological examination of the tumor and lymph node mapping. There were 23 patients (16.5%) in pathological stage I, 36 patients (25.9%) in stage II, 79 patients (56.8%) in stage III, and 1 patient (0.7%) in stage IV.

Survival was examined for each pathological stage (Fig 3). Patients with pathological stage I disease had a 4.3% operative mortality (1/23) and a 3-year survival of 45% (median survival, 1.88 years). Patients with pathological stage II disease had an operative mortality of 2.8% (1/36) and a 3-year survival of 47% (median survival, 2.87 years). Patients with pathological stage III disease had an operative mortality of 6.3% (5/79) and a 3-year survival of 24% (median survival, 1.17 years).

Survival and Perioperative Pulmonary Function

We examined the value of pulmonary function tests as a predictor of operative mortality and survival. Risk factors examined for operative mortality and survival after pneumonectomy included preoperative FVC of 2.13 L or less, preoperative FEV_1 of 1.65 L or less, percent predicted FVC of 64% or less, and percent predicted FEV_1 of 65% or less. In addition, predicted postoperative FVC of 1.31 L or less, predicted postoperative FEV_1 of 0.89 L or less, predicted postoperative percent predicted FVC of 41% or less, and

Table 1. Summary of Data on Patients With an Operative Death[a]

Patient No.	Age (yr)	Stage		Preoperative				Xenon 133 % Ventilation	Predicted Postoperative				Initial Complications	Days Until Death	Cause of Death
		Clinical	Pathological	FEV_1 (L)	% Predicted	FVC (L)	% Predicted		FEV_1 (L)	% Predicted	FVC (L)	% Predicted			
1	58	III	III	3.16	95	4.86	88	ND	. . .	. . .	. . .	. . .	Cardiac arrest	7	Pneumonia, ARF
2	58	III	III	2.67	76	3.96	78	53	1.42	40	2.04	41	Cardiac arrest	12	Pneumonia
3	72	III	III	2.52	85	3.28	84	54	1.36	46	1.74	45	Cardiac arrest	4	Pneumonia
4	45	III	III	2.30	65	3.60	77	52	1.20	34	1.87	40	BPF	22	BPF, ARF, sepsis
5	67	III	II	1.86	58	3.04	64	73	1.35	42	2.21	47	Pneumonia	8	Pneumonia, MOF
6	65	III	III	1.09	47	1.60	55	82	0.89	38	1.31	45	MI	6	Pneumonia
7	55	III	I	1.25	52	1.68	57	59	0.73	31	0.99	34	Pneumonia	10	Pneumonia

[a] There were no deaths in the operating room, and all patients had a right pneumonectomy.

ARF = acute renal failure; BPF = bronchopleural fistula; FEV_1 = forced expiratory volume in 1 second; FVC = forced vital capacity; MI = myocardial infarction; MOF = multiorgan system failure; ND = not done.

predicted postoperative percent predicted FEV_1 of 34% or less were examined. Xenon 133 ventilation/perfusion scans were used to predict postoperative pulmonary function in the noninvolved lung. Actuarial patient survival was examined as a function of perioperative pulmonary function and clinical and pathological stage.

Survival by Clinical Stage and Perioperative Pulmonary Function

There were no operative deaths among patients in clinical stage I or II. The operative mortality among clinical stage III patients was 7.8% (7/90), and death occurred in 20% (2/10) of patients at increased risk (one or more factors as previously described) compared with 6.25% (5/80) of other patients (see Table 1).

Median survival for clinical stage III patients with a preoperative FVC of 2.13 L or less was 0.1 year (n = 6) compared with 1.32 years for those with a preoperative FVC of more than 2.13 L (n = 84) (p = 0.002) (Fig 4A). Median survival for clinical stage III patients with a preoperative FEV_1 of 1.65 L or less was 0.59 year compared with 1.44 years for other clinical stage III patients (p = 0.003) (Fig 4B). No patient in clinical stage III with a percent predicted FVC of 64% or less survived beyond 1.3 years compared with a 30% survival at 3 years for all other clinical stage III patients (p = 0.0001) (Fig 5).

No clinical stage III patient with a predicted postoperative FVC of 1.31 L or less survived beyond 3.1 years compared with a 28% 3-year survival for other patients in clinical stage III (p = 0.07) (Fig 6). Finally, patients in clinical stage III with a predicted postoperative FEV_1 of 0.89 L or less had a 3-year survival of 10% (1 patient at risk) compared with a 28% 3-year survival for other clinical stage III patients (p = not significant).

There were no significant differences in survival after pneumonectomy between groups for the other risk factors examined in clinical stage III patients.

Survival by Pathological Stage and Perioperative Pulmonary Function

Pathological staging was accomplished at operation by lymph node mapping using established criteria [12]. Good-risk patients in pathological stage III had a 1.25% 30-day mortality and a 25% 5-year survival. Median survival by pathological stage was similar to that by clinical stage for groups with comparable pulmonary function.

Median survival for pathological stage III patients with a preoperative FVC of 2.13 L or less was 0.38 year compared with 1.25 years for those with a preoperative FVC of more than 2.13 L (p = not significant). Median survival for pathological stage III patients with a preoperative FEV_1 of 1.65 L or less was 0.70 year compared with 1.27 years for other pathological stage III patients (p = not significant). There were no survivors beyond 3.1 years for pathological stage III patients with an FVC of 2.13 L or less or an FEV_1 of 1.65 L or less.

The percent predicted FVC of 64% or less (preoperative) was associated with decreased survival after pneumonectomy. No patient in pathological stage III with a percent predicted FVC of 64% or less survived beyond 1.3 years

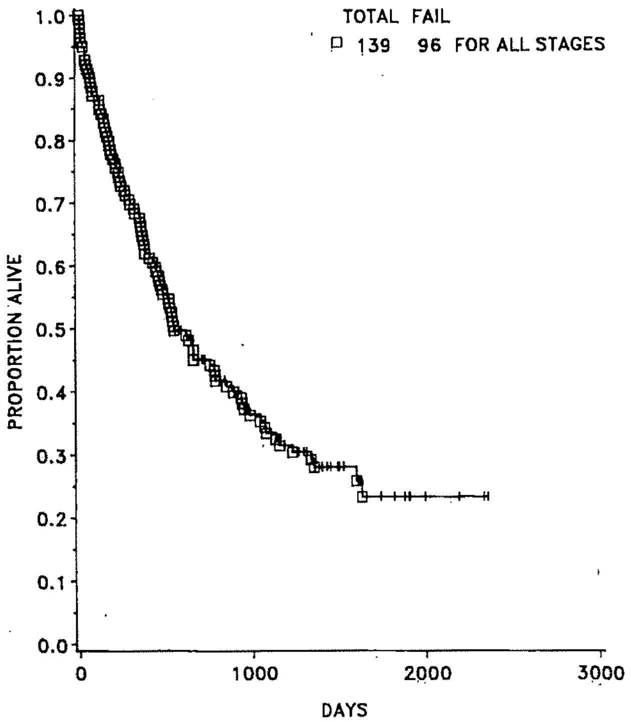

Fig 1. Overall actuarial survival of patients undergoing pneumonectomy for primary carcinoma of the lung.

compared with a 26% survival at 3 years for the other pathological stage III patients ($p = 0.009$).

No patient in pathological stage III with a predicted postoperative FVC of 1.31 L or less or a predicted postoperative FEV_1 of 0.89 L or less survived beyond 3.1 years (p = not significant).

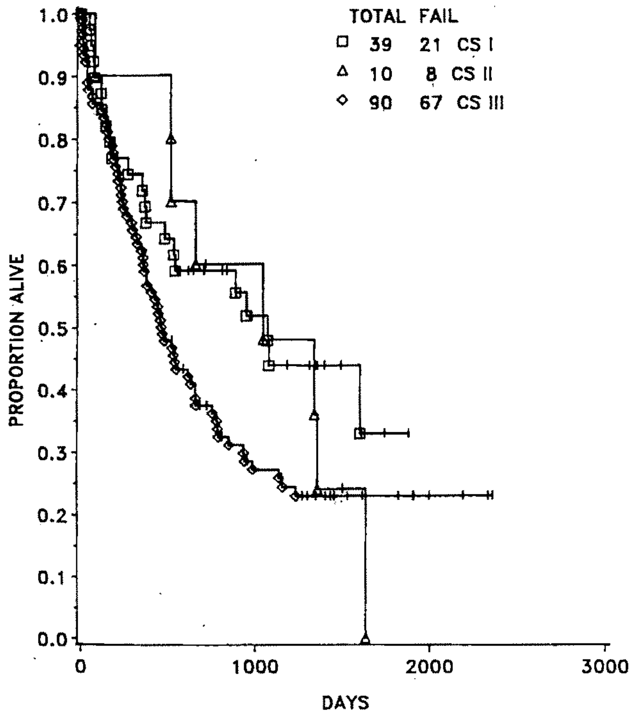

Fig 2. Actuarial survival of patients with clinical stage (CS) I, II, or III primary carcinoma of the lung.

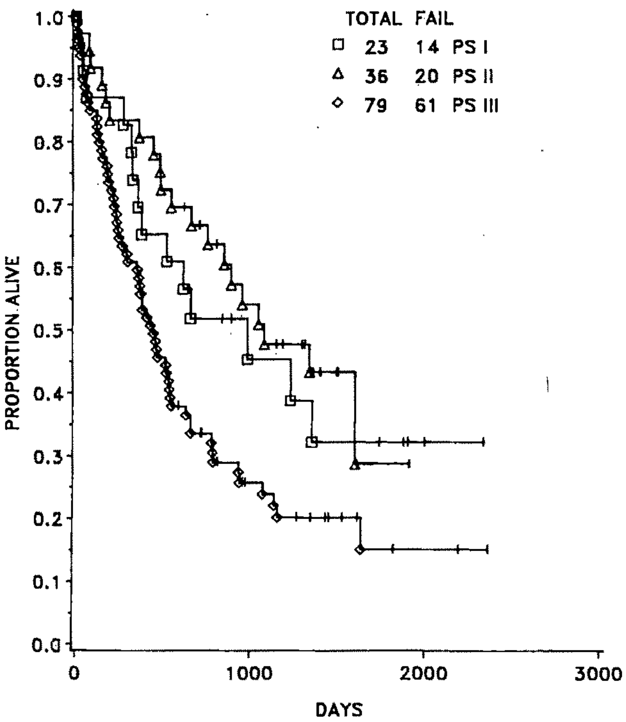

Fig 3. Actuarial survival of patients with pathological stage (PS) I, II, or III primary carcinoma of the lung (based on histological examination of the resected specimen and mediastinal lymph node dissection). One patient (not shown) was found to have pathological stage IV disease and survived for 159 days after pneumonectomy.

Comment

Pneumonectomy for the treatment of lung cancer carries the highest mortality rate among pulmonary resections. The recognized value of this operation as a curative procedure for select patients with bronchogenic carcinoma has led to the continued development of better methods to evaluate these patients preoperatively to identify those patients who would benefit from pneumonectomy and those patients who would be at increased risk from operation.

Appropriate screening of patients before pneumonectomy included a thorough evaluation for metastatic disease, pulmonary function assessment by spirometry, and cardiac evaluation for patients with potential cardiac dysfunction. If pulmonary function by spirometry was marginal, xenon 133 ventilation/perfusion scans were used to predict postoperative lung function in the noninvolved lung after pneumonectomy. Patients with compromised ventilation to the involved lung might have sufficient residual function in the noninvolved lung, and pneumonectomy could be performed with hope of success.

There has been considerable debate over the value of pulmonary resection for clinical stage III bronchogenic carcinoma [17–19]. Often the presence of enlarged mediastinal nodes on computed tomographic scans of the chest has prompted a diagnosis of "unresectable cancer" and eliminated consideration of surgical intervention without further evaluation. Clinical staging is often unreliable, and therapeutic decisions should not be based on clinical

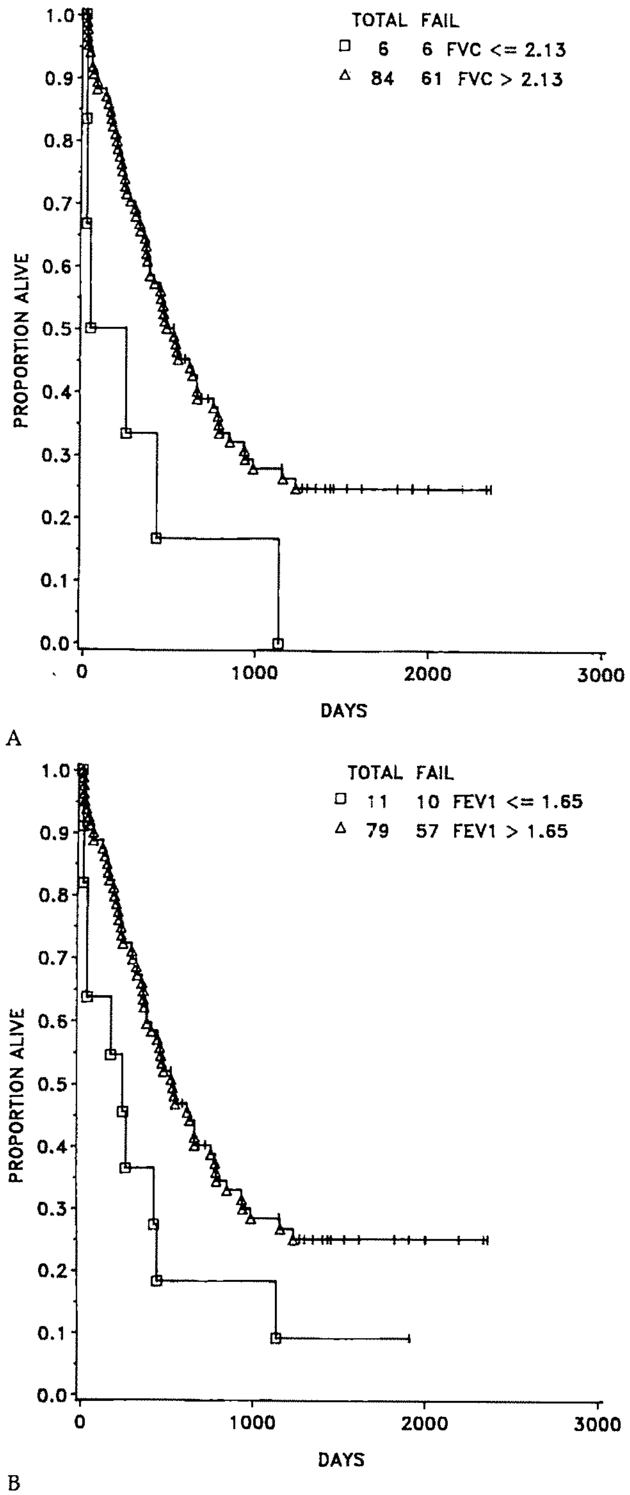

Fig 4. (A) Actuarial survival of patients with clinical stage III lung cancer based on preoperative forced vital capacity (FVC) *in liters* (p = *0.023). (B) Actuarial survival of patients with clinical stage III lung cancer based on preoperative forced expiratory volume in 1 second in liters* (FEV$_1$) (p = *0.034).*

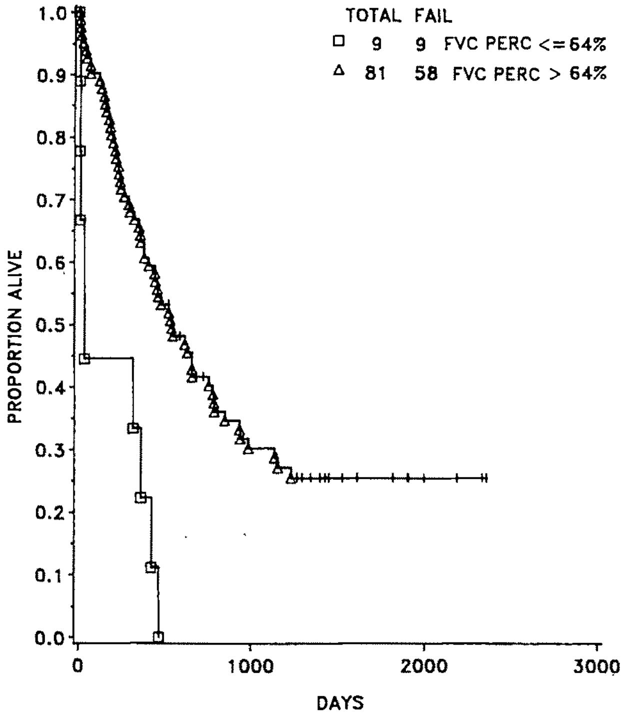

Fig 5. Actuarial survival of patients with clinical stage III lung cancer based on percent predicted forced vital capacity (FVC PERC) *of the noninvolved lung* (p = *0.0001). The percentage of FVC was based on standard values.*

stage alone. We have shown that patients with primary bronchogenic carcinoma, clinical stage III and pathological stage III disease, and good pulmonary function will have an actuarial 3-year survival greater than 25%. Operative mortality was low, and some patients achieved long-term survival.

A recent study from our center [11] identified a group of factors influencing perioperative morbidity and mortality

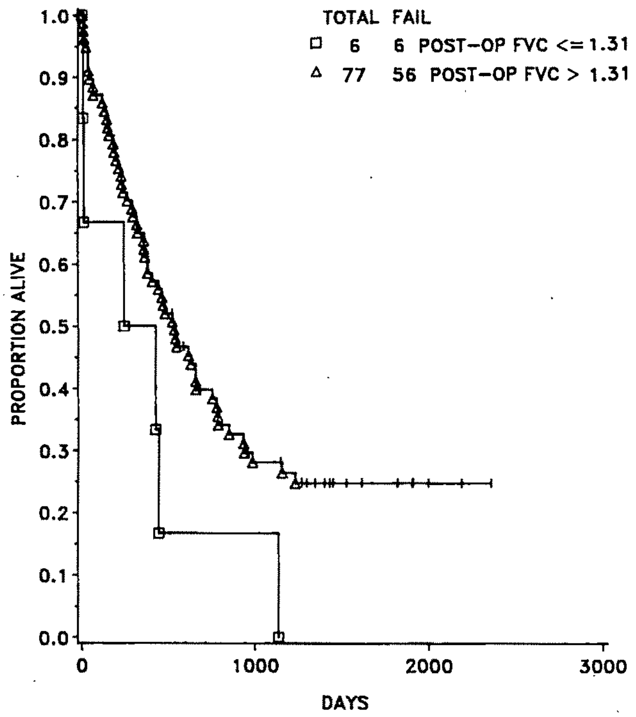

Fig 6. Actuarial survival of patients with clinical stage III lung cancer based on predicted postoperative forced vital capacity (POST-OP FVC) *of the noninvolved lung* (p = *0.07).*

in patients undergoing pneumonectomy. Factors associated with higher risk included extent of resection, right pneumonectomy, and impaired pulmonary function.

The risks of pneumonectomy in clinical stage III patients can be assessed before operation by various pulmonary function studies. Simple spirometry combined with xenon ventilation/perfusion scans can be an effective tool to assist the surgeon in assessing operative risk. Clinical stage III patients with a preoperative FVC of greater than 2.13 L, an FEV_1 of greater than 1.65 L, a percent predicted FVC of more than 64%, a predicted postoperative FVC of greater than 1.31 L, or a predicted postoperative FEV_1 of more than 0.89 L have less risk associated with pneumonectomy. Associated operative mortality is lessened and long-term survival enhanced in patients with these findings.

Patients in pathological stage III with a preoperative FVC of 2.13 L or less, a preoperative FEV_1 of 1.65 L or less, a percent predicted FVC of 64% or less, a predicted postoperative FVC of 1.31 L or less, or a predicted postoperative FEV_1 of 0.89 L or less did not survive beyond 3.1 years.

Patients with clinical stage III disease will benefit from resection if a complete resection can be accomplished. Patients who are in clinical stage III and are considered a good risk based on preoperative and postoperative predicted pulmonary function have a significant improvement in overall survival compared with patients at high risk based on these criteria.

No deaths occurred among patients in clinical stage I or II. Seven deaths occurred in patients who were in clinical stage III. Those patients considered at increased risk had an operative mortality of 20% compared with 6% for other patients. Clinical stage III patients with an FVC greater than 2.13 L and an FEV_1 greater than 1.65 L had an overall operative mortality (5/80, 6.25%) similar to that reported for patients having pneumonectomy by the Lung Cancer Study Group [3] (44/569, 7.7%). If we consider the differences in operative mortality and long-term survival between these two groups of patients within the same clinical stage, it is evident that a group of patients can be identified whose overall survival can be improved by surgical intervention.

Exclusion of patients considered to have clinical stage III lung cancer should be based on associated risk factors including diminished pulmonary function, extent of resection, and overall cardiac status. Although we cannot recommend stringent criteria for operability, these guidelines may assist the surgeon in his or her evaluation of the patient's suitability for pneumonectomy.

In conclusion, patients with clinical stage III disease undergoing pneumonectomy have an actuarial 3-year survival of 28%. Those patients with clinical stage III disease and good pulmonary function had better survival (23% at 5 years) than clinical stage III patients with poor pulmonary function (no survivors beyond 3.1 years). No single variable enables the clinician to predict outcome, particularly death, after pneumonectomy, and patients should not be excluded from pneumonectomy on the basis of any single criterion. Rather, the use of preoperative studies including predicted postoperative pulmonary function by xenon 133 might assist the surgeon in better selecting patients who will potentially benefit from pneumonectomy.

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DISCUSSION

DR JOHN P. CLARKE (Virginia Beach, VA): My associates and I have been using the split-function lung predicted values for a good while and trying to correlate them. I enjoyed your paper; I think it was well done, and I think it was valuable. However, one self-evident factor is that if patients smoke right up to the time that these predicted values are obtained, you really can expect those patients to have a postoperative course that is quite a bit better than the patients with chronic obstructive pulmonary disease who have not smoked for 5 years and have the same values. For instance, a person who has a predicted postoperative FEV_1 of 0.8 L and is a heavy smoker right up to the time of operation is more likely to have long-term survival and tolerance if he or she stops smoking than the chronically ill patient who has stopped smoking and has the same FEV_1.

MR MARK V. BRAIMBRIDGE (London, United Kingdom): I was very interested in the last statement in your conclusion that the predicted values might help the surgeon. When I was working at the Brompton Hospital, my colleagues and I used the stair test in this respect. If the patient reached the first floor, he could survive a segmental resection; if he reached the second floor, a lobectomy; and if he reached the third floor, he could tolerate a pneumonectomy. At St. Thomas' Hospital, this seemed a little crude, and we did a lot of the tests that you recommend, particularly to arrive at the predicted FVC. After trying to calculate this many times, because it obviously is quite different when the lung to be removed is collapsed compared with when it is fully aerated, we found that all our predictive tests were no better than the old-fashioned Brompton three-floor test.

How do you arrive at the predicted FVC? As I see it, that clearly is the critical criterion.

DR LEWIS WETSTEIN (Manalapan, NJ): I congratulate you, Dr Putnam, on your results. My associates and I have also been extremely interested in defining preoperative variables that would decrease operative morbidity and mortality among patients having lung resection. In fact, 3 years ago at this meeting, we looked at exercise oxygen consumption [1]. Dr Bechard and I found that despite adequate preoperative static pulmonary function studies in patients who would equate to your non–high-risk group, there remains a population of patients with major operative morbidity and mortality. We demonstrated that patients who could not generate oxygen consumption of more than 10 mL/kg/min have increased morbidity and mortality, again despite normal static pulmonary function criteria.

Therefore, as a follow-up to Mr Braimbridge, my question is, did you look at an exercise modality? I hypothesize that this would explain why your non–high-risk patients did not do well.

DR WILLIAM L. COX, JR (Las Vegas, NV): Over the last few years in my practice, I have observed that hiatal hernias and reflux have developed in patients with left pneumonectomy. This has been demonstrated in almost 80% of the patients by the cineesophagogram and water test. Some of these symptoms postoperatively I have found to be due to the reflux, which I have recently been repairing when I do a left pneumonectomy. I would certainly appreciate knowing if you had any of these problems in your patients who had a left pneumonectomy.

DR PUTNAM: Dr Clarke, our patients are instructed to stop smoking entirely for 2 weeks before operation to optimize their pulmonary function and minimize their convalescence. Ninety-eight percent of patients comply with this request when they are counseled as to the value of not smoking before operation. If patients who smoke have marginal pulmonary function while smoking, then the pulmonary function tests are reevaluated after these 2 weeks after cessation of smoking. When nonsmoking is coupled with a program of progressive walking and incentive spirometry, operative morbidity is dramatically reduced.

Mr Braimbridge, I agree that walking a patient up three flights of stairs does evaluate adequate pulmonary function before pneumonectomy. Although it is inexpensive, is easy to perform, and provides valuable information, it is somewhat subjective and difficult to quantitate. The pulmonary function studies performed on our patients attempted to quantitate preoperative and predicted postoperative pulmonary function. The best single test for predicting operative morbidity and mortality has not been determined and may be a combination of studies.

The predicted FVC was obtained by a xenon 133 ventilation/perfusion scan. The predicted FVC was calculated by multiplying the percentage of ventilation in the noninvolved lung times the preoperative FVC. This is the "predicted" postoperative FVC.

Dr Wetstein, exercise oxygen consumption is an accurate measure of physiological cardiopulmonary function and can appropriately define operative risk with high-risk patients having less than 10 mL/kg/min oxygen consumption. This test was not available for the entire study period. Currently we have a prospective study ongoing to evaluate the use of oxygen consumption as an indicator of risk in the thoracic surgical patient.

Dr Cox, symptomatic gastroesophageal reflux with aspiration has not been a problem in our patients after pneumonectomy. In patients with a history of reflux, esophageal manometric and pH testing should be performed before operation and the reflux problem should be corrected. If the left chest is to be explored for pulmonary resection, a floppy Nissen fundoplication can be performed readily with Collis gastroplasty if required.

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Endothelial Cell Thromboxane Production and Its Inhibition by a Calcium-Channel Blocker

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Department of Surgery, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina

Vascular endothelium has been established as a major source of prostacyclin production. Whether endothelial cells are also capable of synthesizing thromboxane A_2 remains controversial. Suppression of such endothelial thromboxane synthesis would be beneficial for short-term patency of vascular grafts. This study examined the production of thromboxane A_2 by endothelial cells and its modulation by the calcium-channel blocker nifedipine in vitro. The results indicate that (1) endothelial cells spontaneously secrete thromboxane A_2, (2) this production can be enhanced severalfold in the presence of arachidonic acid, and (3) the calcium-channel blocker nifedipine significantly inhibits thromboxane production without demonstrable toxicity to the endothelial cells.

(Ann Thorac Surg 1990;49:916–9)

Vascular endothelium keeps a balance between hemocompatibility and hemostasis through several mechanisms. One such mechanism is the thromboxane/prostacyclin system that regulates, in part, the interactions of platelets with the vessel wall. It has been suggested that endothelial cells produce prostacyclin, a potent vasodilator and inhibitor of platelet aggregation [1–3], whereas only platelets produce thromboxane A_2, a powerful platelet aggregator and vasoconstrictor [4–6]. Whether endothelial cells can produce thromboxane A_2 has been a subject of controversy [7–9]. If indeed they do, selective inhibition of thromboxane A_2 production on the endothelial surface without affecting prostacyclin synthesis might reduce thrombotic complications and increase short-term patency of bypass grafts.

The purpose of this study was to determine whether endothelial cells in culture can produce thromboxane A_2 (under baseline and stimulated conditions) and whether this production can be inhibited by a calcium-channel blocker.

Material and Methods

Reagents

Nifedipine (molecular weight, 346.3), in powder form and 99.5% pure, was a gift from Pfizer Laboratories, Groton, CT (lot No. 5E127-04QCS-07). Arachidonic acid (AA) was provided by Dr David Henke, University of North Carolina at Chapel Hill.

Cell Culture

All cell culture reagents were obtained through the tissue culture facility of the Lineberger Cancer Center, Chapel Hill, NC. Rabbit aortic endothelial cells (supplied by Dr Fred C. Jensen, Scripps-Miles, Inc, La Jolla, CA) were maintained in Dulbecco's modified Eagle's medium/Ham's F12, a 1:1 mixture, and antibiotics (penicillin sodium, 100 IU/mL, streptomycin sulfate, 100 μg/mL, and amphotericin B, 2 μg/mL). The culture media were supplemented with 10% fetal calf serum for routine maintenance and passaging of the cultures. However, 48 hours before the conduct of the experiments, 10% platelet-poor plasma was substituted for the calf serum and then reduced to 5% to eliminate the contribution of serum platelets to thromboxane in the media.

Six-well culture plates were kept in a humidified 5% carbon dioxide incubator at 37°C. Identity of the endothelial cells was confirmed by their maintenance of density-dependent growth inhibition after serial passage [10], by positive indirect immunofluorescence staining for von Willebrand factor [11], and by their production of prostacyclin as determined by high-performance liquid chromatography.

Determination of Cell Number, Protein Synthesis, and Viability

For counting, cells were washed twice in phosphate-buffered saline solution to remove any nonadherent cells. Then 1 mL of trypsin–EDTA (ethylenediaminetetraacetic acid) solution (0.1% trypsin with 1 mmol/L of EDTA in Hanks' balanced salt solution) was added to each well and the plates were incubated for ten to 15 minutes at 37°C in a 5% carbon dioxide incubator. Once complete detachment was achieved, a 0.5-mL aliquot of each cell suspension was added to 9.5 mL of isotonic diluent (Fisher Diagnostics, Orangeburg, NY) in plastic counting vials. Samples were counted in triplicate on an Elzone Particle Data cell counter (n = 6 wells/group).

Overall cellular protein synthesis was quantitated by the incorporation of tritium-labeled leucine by monolayers of endothelial cells over an eight-hour incubation

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period. The rate of protein synthesis is expressed as counts per minute per 1,000 endothelial cells.

Cell viability was determined by the dye-exclusion method using 0.4% trypan blue and a hemocytometer.

Thromboxane Assay

Levels of thromboxane B_2, the stable hydrolysis product of thromboxane A_2, were determined in all samples by radioimmunoassay using a commercially available kit (New England Nuclear, Boston, MA). Levels were normalized for number of endothelial cells and reported as picograms per million cells.

Experimental Setup

Confluent monolayers of endothelial cells were divided into three groups of 6 wells each. The control group was incubated in culture media (Dulbecco's modified Eagle's medium/Ham's F12) with 5% platelet-poor plasma for 48 hours. The two experimental groups were incubated in identical media under the same conditions with the addition of low-dose nifedipine, 1,000 ng/mL, and high-dose nifedipine, 10,000 ng/mL. The conditioned media from each of the 18 wells were then collected and centrifuged at 800 *g* for 20 minutes to remove cellular debris. They were then assayed for thromboxane as already described. Samples of unconditioned media from all three groups were also assayed for thromboxane and were found to contain only negligible amounts of the metabolite.

In another experiment, the control group ($n = 5$ wells) was incubated in culture media without serum for one hour. The experimental group ($n = 9$ wells) was treated identically, but with the addition of AA to achieve a final concentration of 10 mmol/L. The conditioned media were then collected and processed as already described.

Statistical Analysis

All results were analyzed for significance with the unpaired *t* test.

Results

The effect of AA stimulation on the endothelial cell production of thromboxane is illustrated in Figure 1. There was a 3.85-fold increase in the amount of thromboxane produced when cells were exposed to AA compared with the control group ($p = 0.0001$). This confirms that endothelial cells can spontaneously produce thromboxane and shows that this baseline production can be amplified with AA stimulation.

There was a significant reduction in the baseline production of thromboxane with both low-dose (49%) ($p = 0.04$) and high-dose (59%) ($p = 0.01$) nifedipine (Fig 2).

The effect of high-dose nifedipine on overall cellular protein synthesis is shown in Figure 3. There was no significant difference between the control and nifedipine groups in their rates of protein synthesis. Cell viability, as determined by dye exclusion, was similar for both groups.

Comment

"A membrane as simple as any that is ever met within the body" was Virchow's description of what is now recognized as an active organ of tremendous importance in most biological and homeostatic processes: the endothelium. Once dismissed as just a passive interface between the bloodstream and the tissues, the endothelium is increasingly being recognized as an important modulator of such diverse processes as coagulation, platelet activation, vascular tone, wound healing, leukocyte migration, atherogenesis, and tumor invasion. This is through the production of numerous biologically active substances, which include enzymes, vasoactive substances, mitogens, hemostatic and fibrinolytic agents, chemotactic factors, and matrix proteins.

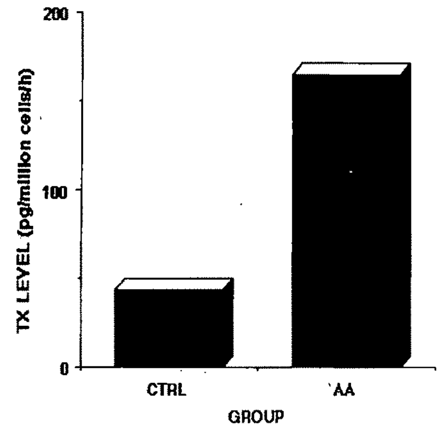

Fig 1. Effect of arachidonic acid (AA) stimulation on the endothelial cell production of thromboxane (TX). Average thromboxane levels were 43.0 pg/million cells/h for the control group (CTRL) ($n = 6$ wells) and 165.5 pg/million cells/h for the AA group ($n = 9$ wells) ($p = 0.0001$.)

The eicosanoids form a family of endothelial products affecting vascular tone and blood vessel/platelet interactions. Eicosanoid metabolism starts with the release of AA from membrane phospholipids after the activation of phospholipases. Arachidonic acid is then acted upon by a cyclooxygenase and a hydroperoxidase that convert it to prostaglandin G_2 and then prostaglandin H_2. Beyond this

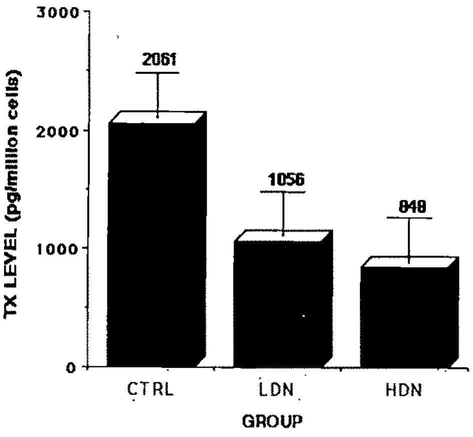

Fig 2. Effect of nifedipine, 1,000 ng/mL and 10,000 ng/mL, on spontaneous thromboxane (TX) production. Data shown are means ($n = 6$ wells per group). (CTRL = control; HDN = high-dose nifedipine; LDN = low-dose nifedipine.)

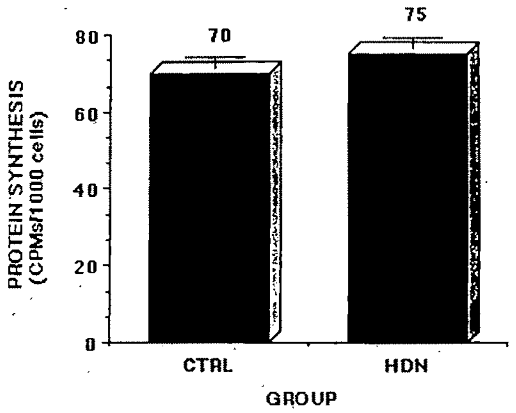

Fig 3. *Protein synthesis in the absence and presence of nifedipine. Data shown are means (n = 6 wells per group). There was no significant difference (p = 0.3).* (CTRL = *control;* HDN = *high-dose nifedipine, 10,000 ng/mL.)*

point in the pathway, cells show selectivity, depending on their nature and function, in their metabolism of prostaglandin H_2 to different biologically active prostanoids.

It has been demonstrated [7, 12] that under normal conditions, the prostacyclin to thromboxane ratio is in favor of prostacyclin 5–10:1. This is responsible, at least in part, for maintaining endothelial thromboresistance and hemocompatibility [13]. This balance, however, is easily altered in situations of endothelial injury or in certain pathological states. Pharmacological inhibition of thromboxane A_2 synthesis, in a selective fashion, would be desirable in regard to prevention of thrombosis. However, currently used agents (cyclooxygenase inhibitors) cause nonspecific inhibition of the entire prostaglandin synthetic pathway and thus reduce prostacyclin synthesis as well (Fig 4).

Our results clearly demonstrate the production of thromboxane, both spontaneously and after AA stimulation, by endothelial cells in culture. Further, the calcium-channel blocker nifedipine significantly inhibited thromboxane production but did not have any grossly toxic effects on the cells or their function. A hypothetical site for this observed effect is depicted in Figure 5. Previous work from our laboratory [14] has also shown nifedipine to cause a beneficial inhibition of endothelial mitogen production. These two theoretically desirable effects seen in vitro, coupled with the limitations of the "shotgun" inhibition of prostaglandin synthesis obtained with current cyclooxygenase inhibitors [13, 15], might warrant clinical investigation of a potential role for calcium-channel blockers in modulating thrombosis and limiting failure of vascular grafts. A natural extension of this work, that is, the study of the effect of nifedipine on prostacyclin synthesis and on prostacyclin to thromboxane ratios, is currently underway.

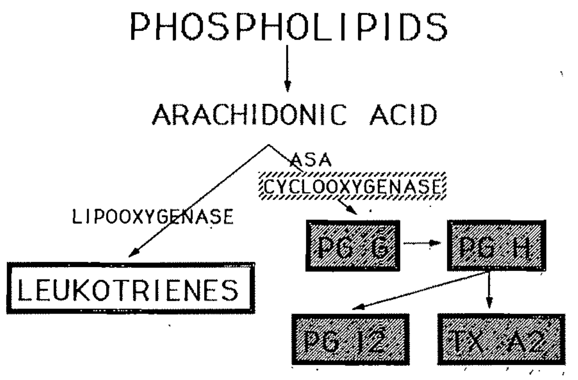

Fig 4. *Arachidonic acid metabolism with site of action of aspirin* (ASA). *(*PG G = *prostaglandin G;* PG H = *prostaglandin H;* PG I2 = prostacyclin; TX A2 = *thromboxane A_2.)*

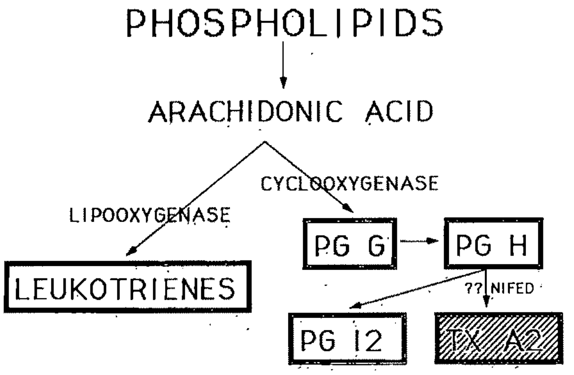

Fig 5. *Hypothesized site of observed nifedipine* (NIFED) *effect on arachidonic acid metabolism.* (PG G = *prostaglandin G;* PG H = *prostaglandin H;* PG I2 = *prostacyclin;* TX A2 = *thromboxane A_2;* ?? = *this is only a hypothesized effect.)*

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The Thirty-seventh Annual Meeting of the Southern Thoracic Surgical Association will be held at the Hyatt Regency Cerromar Beach, Puerto Rico, November 8–10, 1990. There will be a $155 registration fee for nonmember physicians except for guest speakers, authors and coauthors on the program, and residents. There will be a $50 registration fee for attendees of the Postgraduate Course, which will be held the morning of Thursday, November 8, 1990. The Postgraduate Course will provide in-depth coverage of thoracic surgical topics selected primarily as a means to enhance and broaden the knowledge of practicing thoracic and cardiac surgeons.

Members wishing to participate in the Scientific Program should submit an original abstract and one copy by May 15, 1990, to Donald C. Watson, Jr, MD, Program Chairman, Southern Thoracic Surgical Association, 111 East Wacker Drive, Chicago, IL 60601. Abstracts must be submitted on the Southern Thoracic Surgical Association abstract submission form. These forms may be obtained from the Association's office. All slides used during the presentation must be 35 mm. Manuscripts of accepted papers must be submitted to *The Annals of Thoracic Surgery* prior to the 1990 meeting or to the Secretary-Treasurer at the opening of the Scientific Session.

Applications for membership should be completed by July 1, 1990, and forwarded to Lenox Baker, Jr, MD, Membership Committee Chairman, Southern Thoracic Surgical Association, 111 East Wacker Drive, Chicago, IL 60601.

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Incidence and Risk of Reintervention After Coarctation Repair

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We examined the need for intervention after coarctation repair in a retrospective study of 197 procedures performed between 1967 and 1989. Reintervention was required in 23 patients. No technique of coarctation repair was free from complications. Although there were only two stenoses in the group receiving Dacron patch angioplasty, only seven of these procedures were performed in children under the age of 1 year. The risk of stenosis was inversely correlated to the age at primary repair, with children less than 1 year old being at greater risk than those more than 1 year of age ($p < 0.05$). Subclavian flap angioplasty had a lower risk of reoperation than end-to-end anastomosis ($p < 0.02$). Formation of true aneurysms was confined to the Dacron patch angioplasty group. The morbidity and mortality for reintervention was low in all groups, with only one procedure-related death and no incidence of paraplegia. Although no technique is free from risk, subclavian flap angioplasty leads to fewer reinterventions in younger patients.

(Ann Thorac Surg 1990;49:920–6)

The operative treatment of coarctation of the aorta has resulted in improved survival of patients who would have died of cardiovascular complications of persistent hypertension. However, the long-term results of operation for coarctation of the aorta have been confused by the multiplicity of procedures performed and by the associated late complications necessitating additional interventions. The purpose of this retrospective analysis was to determine the incidence of reintervention after initial coarctation operations at the University of Virginia and to determine the risk of reintervention.

Material and Methods

One hundred ninety-seven operations for repair of coarctation of the aorta were performed at the University of Virginia between 1967 and 1989. Four types of operations were done. The most frequently performed operation was coarctectomy and end-to-end anastomosis. Subclavian flap angioplasty was used primarily for infants and young children. Dacron patch angioplasty of the coarctate area was generally performed in older children and infrequently in children less than 1 year of age. Finally, interposition grafts were used in a small number of patients.

Surgical Techniques

END-TO-END ANASTOMOSIS (Fig 1). The aorta was isolated with clamps, and the coarctate segment was excised after the ligamentum arteriosum or patent ductus was ligated and transected. An end-to-end anastomosis was performed using a running suture for the back row. Interrupted sutures were then used for the anterior row of the anastomosis.

SUBCLAVIAN FLAP ANGIOPLASTY (Fig 2). The aorta was clamped as already mentioned. The subclavian artery was transected at the takeoff of the vertebral and internal mammary arteries. These arteries were occasionally individually ligated if more length was needed. A vertical incision was made distal to the coarctation area and carried proximally to the subclavian artery. The subclavian artery was used as a flap and anastomosed over the coarctate area as a patch with a running monofilament suture. The posterior ridge was excised if it was prominent.

DACRON PATCH ANGIOPLASTY (Fig 3). The aorta was isolated as already described. Arteriotomy was performed through the coarctate segment. The posterior ridge was excised if it was prominent. A preclotted knitted Dacron graft was sutured into place with a running monofilament suture.

INTERPOSITION GRAFTING. After isolation, the coarctate segment was excised. It was replaced with an interposition graft of knitted Dacron.

Follow-up

The patients were seen postoperatively on a yearly basis. Standard sphygmomanometric pulse-volume recordings and, more recently, Doppler echocardiography were used to measure gradients across the coarctation segment [1]. If hypertension developed or there was a gradient greater than or equal to 30 mm Hg across the coarctation segment, repeat angiography and reintervention were performed. Reintervention included patch angioplasty, inter-

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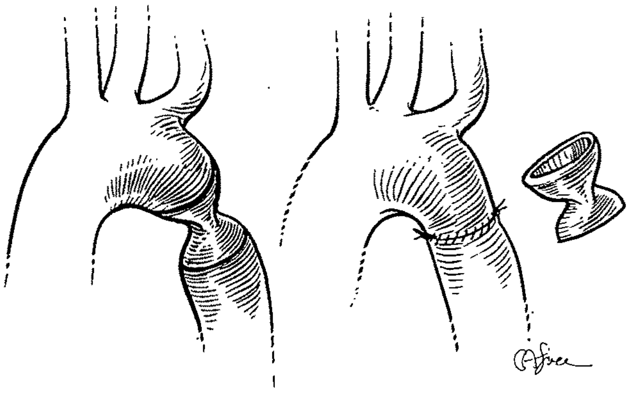

Fig 1. *End-to-end anastomosis.*

position grafting (particularly for aneurysms), and balloon angioplasty if applicable. Neither shunts nor cardiopulmonary bypass was used for any of the primary or secondary procedures.

Statistical Analysis

Statistical analyses were performed using analysis of variance for comparisons among groups. Paired data were compared using Student's *t*-test. Significance was achieved when p was less than 0.05.

Results

Primary Operations

Seventy-six patients underwent end-to-end anastomosis after coarctectomy (Table 1). There were three operative deaths, all related to other cardiovascular conditions. Fifteen survivors (21%) required reoperation or balloon dilation. All had adequate reduction of the gradient. No aneurysms occurred in this group.

Fifty-eight patients underwent subclavian flap angioplasty (see Table 1). There were three operative deaths (5.2%), all related to other cardiac conditions. Paraplegia developed in 1 infant. No patient experienced arm ischemia. Five patients required reintervention for recurrent coarctation (9.1%). Two of the stenoses were in the arch proximal to the repair. No aneurysms developed in this group.

Fifty-six patients underwent Dacron patch angioplasty (see Table 1). There were no operative deaths. Two patients (3.6%) had recurrent coarctation necessitating a second procedure. However, aneurysms developed in 9 patients. All were true aneurysms, and four have been repaired.

Seven patients had interposition grafting (see Table 1). There were no operative deaths. One patient had restenosis necessitating reintervention. A false aneurysm developed in 1 patient and was repaired.

Aneurysms

No aneurysms occurred in patients undergoing either end-to-end anastomosis or subclavian flap angioplasty. In the other two groups, aneurysms developed in 10 patients. Nine of the aneurysms were thought to be true aneurysms, and one was considered a false aneurysm related to infection after interposition grafting. Four of the true aneurysms were treated surgically (Table 2). All

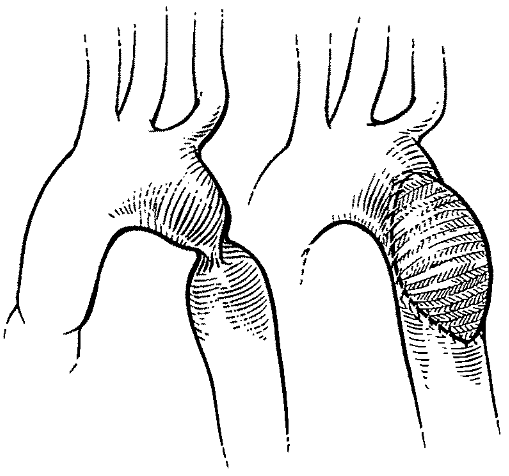

Fig 3. *Dacron patch angioplasty.*

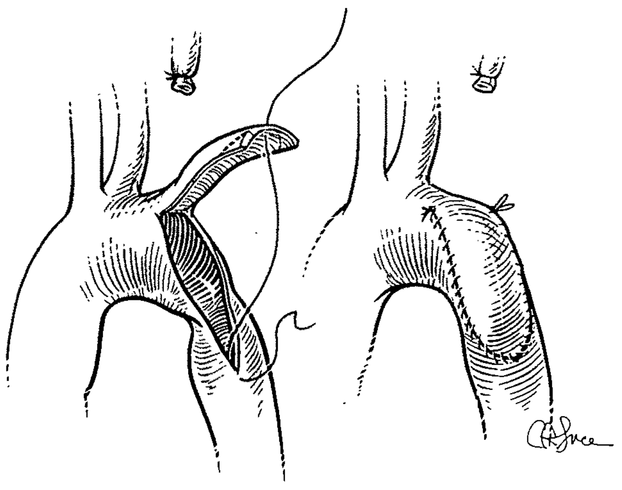

Fig 2. *Subclavian flap angioplasty.*

Table 1. Summary of Primary Surgical Techniques and Results

Technique	No. of Patients	Operative Deaths	Reintervention for Coarctation	Aneurysm Formation
End-to-end anastomosis	76	3	15	0
Subclavian flap	58	3	5	0
Dacron patch	56	0	2	9
Interposition graft	7	0	1	1
Total	197	6	23	10[a]

[a] Five required repair, four in the Dacron patch group and the other in the interposition graft group.

Table 2. Summary of Data on Aneurysms Requiring Repair

Primary Procedure	Age (yr)	Type of Aneurysm	Repair	Age (yr)	Clamp Time (min)	Complication
Patch	14	True	Dacron tube	18	30	Vagus injury
Patch	3	True + proximal recoarctation	Dacron tube + reimplantation of subclavian	12	31	None
End-to-end anastomosis (6/73); patch (1/79)	1 6	True	Dacron tube	15	19	None
Patch	6	True	Dacron tube	7	29	None
Aortic graft	1	False	Dacron tube	11	37	None

reoperations were performed using simple clamping and excision without shunts or bypass. The clamp times were relatively short, and no paraplegia or neurological injury occurred postoperatively. Theories regarding the etiology of these aneurysms after patch angioplasty have been discussed previously [2, 3].

None of the four types of operation was free from complications. The risk of stenosis was inversely related to the patient's age at primary repair. Children less than 1 year of age were at greater risk than children more than 1 year of age ($p < 0.05$) (Table 3). Subclavian flap angioplasty was associated with a lower risk of reoperation compared with end-to-end anastomosis ($p < 0.02$). However, the morbidity and mortality of reintervention was low for all four types of primary operation. There was only one operative death (4.3%) and no incidence of paraplegia (Table 4).

Comment

After the description of surgical intervention for coarctation of the aorta by Crafoord and Nylin [4], resection and end-to-end anastomosis was the standard operation. The long-term results of this therapy were excellent for patients who were operated on after infancy, and there was a low incidence of recoarctation [5]. Infants, however, had a relatively high incidence of restenosis after end-to-end resection, as noted by Sade and colleagues [6]. They found that end-to-end anastomosis after coarctation resection resulted in a significantly higher incidence of restenosis compared with the patch graft aortoplasty technique, originally described by Vosschulte [7]. Sade and coworkers concluded that patch graft aortoplasty was a more effective technique than end-to-end anastomosis for reducing the proximal aortic pressure in infants and small children.

The patch technique was used commonly at many medical centers, including our own, until the appearance of reports of late aneurysm formation. Bergdahl and Ljungqvist [8] reported this complication in 1980. Their study included 1 patient who died of a ruptured aneurysm 17 years after successful coarctation repair. Others [9], including ourselves [2, 3], have confirmed the occurrence of this complication, which has a reported incidence of 5% to 38% after patch angioplasty. It is still not certain why these aneurysms develop. Many authors agree that the posterior wall of the coarctation segment, which is necessarily left in place, probably contributes to the problem. The situation can be particularly difficult if the fibrous membrane of the aorta is resected, as this could lead to a higher incidence of aneurysm formation [10, 11].

The subclavian flap technique was introduced in 1966 by Waldhausen and Nahrwold [12] and has continued to gain acceptance, particularly as used in infants and small children. As dissatisfaction grew with the end-to-end anastomosis technique, the subclavian flap technique replaced it in most centers, including our own [13]. The subclavian flap was noted to have growth potential [14] and was thought to be safer than end-to-end resection in infants [14–16]. There was a lower recurrence rate when the subclavian flap was used.

Even the subclavian flap technique, however, has not been free from complications. Earhardt and Walker [17] noted an 18% recurrence rate after subclavian flap angioplasty in infants. Sanchez and associates [18] suggested that the periductal tissues were responsible for recoarctation after subclavian flap angioplasty and reported a 23% recoarctation rate using this technique. Martin and co-

Table 3. Incidence of Restenosis by Age at Primary Procedure[a]

Age (yr)	End-to-End Anastomosis	Subclavian Flap Angioplasty	Dacron Angioplasty	Interposition Grafting
≤1	13/44[b]	5/41[b]	0/7	0/0
>1	3/32	0/17	2/49	1/7
Total	16/76	5/58	2/56	1/7

[a] Data are shown as the number of patients with restenosis/number of procedures.

[b] Significance: $p < 0.05$ for age comparisons within this procedure.

Table 4. Summary of Data on 23 Patients With Recurrent Coarctation

Age at Primary Procedure (mo)	Primary Procedure	Gradient Before Reintervention (mm Hg)	Reintervention Procedure	Site of Obstruction	Result and Date of Last Follow-up
78	ETE	16	Patch	Previous anastomosis site	Alive and well, 12/8/88
1	ETE	36	Patch	Previous anastomosis site	Alive and well, 12/15/88
84	ETE	30	Patch	Previous anastomosis site	Alive and well, 7/3/89
0.5	ETE	NA	Patch	Previous anastomosis site	Alive and well, 4/24/89
1.25	ETE	NA	SF	Previous anastomosis site	Alive and well, 7/11/88
0.5	ETE	NA	Patch	Previous anastomosis site	Alive and well, 6/89
25	ETE	40	Balloon	Previous anastomosis site	Alive and well, 5/26/84
72	ETE	36	Graft	Hypoplastic arch	Alive and well, 4/18/89
1.0	ETE	36	ETE redo	Previous anastomosis site	Alive and well, 6/5/89
2.0	ETE	40	Patch	Previous anastomosis site	Alive and well, 9/25/85
1.0	ETE	28	Patch	Previous anastomosis site	Alive and well, 5/22/89
0.5	ETE	50	Patch	Previous anastomosis site	Alive and well, 12/8/88
1.0	ETE	30	Patch	Previous anastomosis site	Alive and well, 11/15/86
3.0	ETE	44	Patch	Previous anastomosis site	Alive and well, 10/11/88
5.0	ETE	25	Patch	Distal to left subclavian	Alive and well, 7/25/89
0.5	SF	35	Balloon	SF repair site	Alive and well, 5/22/88
0.5	SF	NA	Graft	Proximal arch	Intraop death
1.0	SF	NA	ETE	Previous repair site	Alive and well, 6/21/89
1.25	SF	62	Balloon	Previous repair site	Alive and well, 6/89
5.0	SF	80	Balloon	Proximal arch	Alive and well, 6/89
102	IG	40	Balloon	Proximal suture line	Alive and well, 6/8/87
84	Patch	30	Patch	Anastomosis site	Alive and well, 7/3/89
		34	Balloon		
5.0	Patch	34	Patch	Distal to left subclavian, proximal end of patch	Alive and well, 7/25/89

ETE = end-to-end anastomosis; IG = interposition graft; NA = not available (catheterization report did not have gradient calculated); Patch = Dacron patch; SF = subclavian flap.

workers [19] also found that aneurysms can occur after subclavian flap angioplasty, although none of the aneurysms had required resection at the time of their report.

We have not seen aneurysm formation after subclavian flap angioplasty. Recent reviews suggest that there is no difference between resection and subclavian flap angioplasty in terms of restenosis. Beekman and colleagues [20] reported a 16% chance of reoperation at 5 years for subclavian flap angioplasty performed in infancy. Cobanoglu and co-workers [21] suggested that an end-to-end anastomosis in infants may, in fact, be the better technique because of a lower reoperation rate. Absorbable monofilament sutures and more extended resections with end-to-end anastomoses have been used by many investigators, particularly for the more extended forms of coarctation in infancy [22–26].

Reoperation for Recurrent Coarctation

Multiple techniques of reoperation for aortic coarctation have been performed. The most common technique we use for recoarctation is patch graft angioplasty with Dacron. Despite our finding that this can lead to development of aortic aneurysm in some instances, it is an excellent technique for recurrent stenosis, particularly after an end-to-end anastomosis. The technique has been well described and is very flexible, particularly with long areas of coarctation [27]. We have also performed subclavian flap procedures after end-to-end anastomoses and occasional interposition grafts that failed, and have found these techniques to be safe. We have not performed bypass of the coarctation areas as described by Jacob and colleagues [28]. Despite substantial adhesions, the operations have been performed with low morbidity; only 1 patient has required reexploration for bleeding postoperatively. Sweeney and associates [29] reported 53 patients requiring reoperation. Twenty-six were treated with patch aortoplasty and the rest, with resection or bypass. Reoperation was successful, there were no deaths, and only 3 patients had residual gradients.

Reoperation for Aneurysm

We have operated on five aneurysms that developed after coarctation repair. Four were true aneurysms, and one was a false aneurysm that formed after an interposition graft was placed. In all situations we used the technique of clamping followed by resection. We have had no incidence of paraplegia in any of the patients having reoperation, including the aneurysm group. Neurological injury has been reported as a complication of coarctation repair [30]. Using evoked potentials, Dasmahapatra and co-workers [31] identified a 26% incidence of reversible ischemia during coarctation repair. The results of Pollock and associates [32] were similar. We believe that as long as the clamp time can be kept short, then it is appropriate to do these repairs without adjuncts such as bypass or shunts. However, this is still an area of controversy.

Balloon Dilation

Transluminal balloon angioplasty has been used for primary and postoperative coarctations. The results of balloon angioplasty for primary coarctation are not uniformly excellent [33]. It has been used very successfully for recurrent coarctation [34]. Hess and associates [35] reported an 80% success rate with this technique when used for recurrent coarctation. In their study, Allen and coworkers [36] found that seven of eight balloon dilations for recurring coarctations were successful. Saul and colleagues [37] had a 90% success rate in a large series of patients with postoperative aortic obstruction. This technique has been an excellent alternative to reoperation for many patients with recurrent coarctation.

All techniques have an incidence of restenosis, particularly when the operation is performed during infancy [38]. In our hands, subclavian flap angioplasty seems to lead to fewer reinterventions, even when performed in infants.

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DISCUSSION

DR THOMAS L. SPRAY (St. Louis, MO): Dr Kron was kind enough to show me a copy of the manuscript before his presentation, and he has a very balanced series of three different types of repair. Considering all the variations in the anatomy of coarctation of the aorta, how would you now manage a patient, an infant rather than an older child, with coarctation? Do you tailor the repair to the type of anatomy seen, do more extended end-to-end resections, or try to use a subclavian flap as your primary repair of choice?

DR KRON: That is an excellent question. We try to use subclavian flap angioplasty as much as we can. We think that is a simple operation and one that is safe to do. But there are infants with very long arch stenoses and hypoplastic arches, and we have done a couple of extended end-to-end anastomoses. Another alternative, particularly if repair of other cardiac conditions is necessary, is to patch the whole area with homograft; that has worked very well. That is the way we have approached it. We try to individualize the operation.

DR RICHARD E. CLARK (President): Dr Kron, when you take down the subclavian, do you tie off the vertebral artery?

DR KRON: We do not make a point of that. I know some think that if you do not, there will be late subclavian steal, but that literature is not clear, and we have not seen that complication.

DR SPRAY: I urge caution about the use of a Dacron graft for repair of recurrent coarctation. We recently had an unfortunate experience with 2 adults who had Dacron graft repair of recoarctation and were seen with exsanguinating aortobronchial fistulas from a false aneurysm that occurred at the site of the Dacron graft anastomosis to the aorta.

Are you using balloon dilation as primary therapy for restenosis, or would you use techniques that would not require Dacron, such as the subclavian flap, as a secondary repair or an end-to-end anastomosis with resection of the recoarctation if a subclavian flap had been used at the first operation?

DR KRON: That is an excellent question. We certainly would try balloon angioplasty first in all children. The experience is good, and the procedure has been safe in our institution; we would use other reconstructions later. We have not seen patch erosion into the airway after coarctation repair. We have seen it after thoracic aneurysm repair, and it is a real concern.

DR SAFUH ATTAR (Baltimore, MD): I have an alternative for these complicated cases, which is based on our long-term experience. My colleagues and I have had 2 cases of paraplegia after recurrent coarctation and repair, one in a young child who had three repairs until he was 20 years old, and the other in a patient with an aneurysm that developed about 7 years after initial coarctation repair. Therefore, we have adopted the policy of avoiding direct attack on the coarctation in children. Instead, we use the bypass procedure, either from the subclavian or other source down to the descending aorta, thereby obviating the serious complication of paraplegia. I offer this solution whenever it is applicable.

DR KRON: I think that is an excellent alternative. We have not had the problem of paraplegia, and I guess we will continue to use a direct approach for now.

DR WILLIAM E. WALKER (Houston, TX): I am not the great hemodynamics expert of the world, but I do not believe blood bounces off Dacron patches and causes aneurysms. I wonder if the cause of all the aneurysms you had was the resection of the posterior ridge. We examined our experience with redo coarctations at Texas Heart Institute 4 years ago, and we had no instance of true aneurysm related to a Dacron patch [1]. I suspect you are making the aorta thin posteriorly.

DR KRON: We had sort of hoped we would get that question. It may be you are not looking hard enough. Three of the aneurysms in our series occurred without resection of the posterior ridge, so I do not think that is the cause.

DR ROBERT A. GUSTAFSON (Morgantown, WV): I agree with your conclusion that subclavian flap angioplasty is very safe in infancy. I think you have shown that the overall risk of intervention in coarctation is low and that the late problems with Dacron patch aortoplasty are important. From your review, what do you think is the ideal operation and what is the ideal age for operation? At my institution, we have found that subclavian flap angioplasty is a very safe operation in infants. In the last 5 years, we have operated on more than 60 patients with coarctation at the time of initial diagnosis, most in early infancy, without any deaths or recurrent coarctation. Should all coarctations be repaired in infancy?

DR KRON: As I said, we think subclavian flap angioplasty is an excellent operation, and we like to use it whenever we can. If you can avoid coarctation repair in infancy, I think you are better off. However, the infants referred to us are usually sick and have heart failure, and we have to repair the coarctation right then. I do not think there is any perfect operation, though. These children have to be followed, and we are still going to see some restenoses, no matter what we do.

DR ROBERT M. SADE (Charleston, SC): I will focus on the issue of aneurysm formation and ask one question. We have not seen an aneurysm in 15 years of following a substantial number of patients with prosthetic patch angioplasty. I do not think one creates aneurysms by resecting aortic wall, and I wonder whether Kron and associates are really seeing as many aneurysms as they think. The angiograms shown certainly revealed an aneurysm: we saw an early postoperative film and a late postoperative film that demonstrated progressive dilatation of the aorta. I, and I believe most surgeons, would have operated on that patient as did Kron and associates.

My co-workers and I were recently referred a patient whose angiogram allegedly documented an aneurysm and were asked to repair it. We restudied the patient, and our angiogram showed no evidence of true aneurysm formation; rather it was a fairly typical picture of a large patch angioplasty of the type we often use. We did not operate on the repaired coarctation. We have had excellent long-term results with patch aortoplasty, have seen very few residual stenoses, and have not yet found an aneurysm.

In how many of the 9 patients reported to have an aneurysm and in how many of the 4 patients operated on for aneurysm was there clear documentation that this was in fact an aneurysm, that there was local aortic enlargement that was expanding over time?

DR KRON: That is a good question. There certainly is a little more dilatation after a patch graft. However, in our group, all were aneurysms. I operated on them, and they were, on average, about 6 cm in size. I do not think you would make a patch that big, and we certainly do not. However, some aneurysms are smaller than that, and if they do not grow, we would not resect them.

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Prostaglandin E_2 Inhibits In Vitro and In Vivo Lymphocyte Responses in Allogeneic Transplantation

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Prostaglandin E_2 (PGE_2) has been shown to have a clear role in the suppression of immune responses after burn and trauma injury. This probably results from inhibition of interleukin-2 production. This study examined the effects of PGE_2 in vivo on the survival of solid-organ allografts and in vitro on the rat allogeneic mixed lymphocyte response. Administration of 16,16-dimethyl prostaglandin E_2 ($DMPGE_2$), a stable analogue of PGE_2, significantly prolonged the survival of heterotopic cardiac allografts from ACI to LBN rats: 10.4 ± 1.7 days versus 5.7 ± 1.1 days (mean ± standard error of the mean) ($p \leq 0.001$). In 1 animal, $DMPGE_2$ apparently led to the induction of long-term tolerance. Mixed lymphocyte cultures using splenocytes from naive LBN and ACI rats to which $DMPGE_2$ was added showed a dose-dependent suppression of the mixed lymphocyte response with concentrations as low as 1×10^{-7} mol/L. Splenocytes harvested from treated animals with functioning but histologically rejecting hearts demonstrated a marked decrease in mixed lymphocyte response to donor (ACI) stimulators compared with naive LBN controls (3,804 ± 603 versus 27,395 ± 2,668 cpm, n = 4), but maintained a normal response to third-party (Wistar Furth) stimulators. We conclude that $DMPGE_2$ suppressed solid-organ allograft rejection, inhibited the allogeneic mixed lymphocyte response, and induced donor-specific in vitro hyporesponsiveness.

(Ann Thorac Surg 1990;49:927–31)

Prostaglandins are now well-recognized modulators of local immune function [1, 2]. The E series prostaglandins have been shown by numerous investigators [3–5] to play a role in the suppression of immune responses after burn or trauma injury. This suppression has been attributed to the inhibition of both interleukin-2 (IL-2) production and IL-2–dependent lymphocyte proliferation [6]. Other IL-2 modulators are currently in wide use for immunosuppression after transplantation. However, prostaglandins, particularly prostaglandin E_2 (PGE_2), have not been thoroughly studied as transplant immunosuppressants. 16,16-dimethyl prostaglandin E_2 ($DMPGE_2$) is a long acting, injectable analogue of PGE_2. In this study we tested the hypothesis that exogenous administration of PGE_2 as $DMPGE_2$ would suppress alloreactivity both in vivo and in vitro and, therefore, prolong solid-organ allograft survival.

Material and Methods

To test this hypothesis, we studied rat mixed lymphocyte cultures in vitro and rat heterotopic heart transplants in vivo. Fully allogeneic cardiac allografts from ACI rats were transplanted into Lewis × Brown Norway F1 (LBN) recipients using a modification of the technique of Ono and Lindsey [7]. The same combination was used for the mixed lymphocyte cultures for both transplanted and naive rats with Wistar Furth (WF) rats serving as third-party stimulators.

Transplantation

The ACI and LBN rats, weighing 100 and 300 g, respectively, were obtained from National Institutes of Health stock and were cared for according to the "Guide for the Care and Use of Laboratory Animals" published by the National Institutes of Health (NIH publication No. 85-23, revised 1985). They were not given food the night before operation, but had free access to water.

A standard ether jar was used to anesthetize both donor and recipient animals initially. Subsequently, the LBN (recipient) rats received intraperitoneal administration of sodium pentobarbital (Richmond Veterinary Supply Company), 1.0 g/mL, at 0.1 mL/100 g to maintain anesthesia. A plastic test tube filled with gauze was used as an ether mask to maintain anesthesia in the ACI (donor) rats.

Recipient rats underwent a midline laparotomy and simultaneous occlusion of both the aorta and the venae cavae with a single partially occluding atraumatic vascular clamp. They were then set aside while the donors were prepared. After the induction of general anesthesia, the donor rats were given 1,000 units of heparin sodium intravenously. After two minutes of circulation, a median sternotomy was performed, the inferior vena cava was divided, and 10 mL of cold NIH cardioplegia was administered through the aortic arch. The heart was then

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rapidly removed using a single 4-0 silk suture to ligate the pulmonary veins and superior vena cava, and was kept cold with topical iced saline solution during transplantation.

End-to-side anastomoses were performed between the donor aorta and the recipient aorta and between the pulmonary arteries and the venae cavae. The average time of ischemia was 30 minutes, and spontaneous systolic function usually began within two minutes after release of the clamp. The animals received no prophylactic antibiotics, and clean, but not sterile, technique was used.

Postoperatively, the rats were put in a warmed cage (a single 75-W bulb placed at a distance compatible with a cage temperature of 29.7°C) and given free access to water and Rat Chow (Ralston Purina). All animals were examined daily by palpation for the presence and strength of graft cardiac impulse. Rats were killed at regular intervals (days 1, 3, and 5 in the control group and days 3, 5, 7, and 9 in the treated group) before rejection for histological analysis.

Treated animals were given DMPGE$_2$, obtained as a generous gift from The Upjohn Company (Kalamazoo, MI). Fresh aliquots of a stock solution (5 mg/mL) of DMPGE$_2$ kept at −20°C were diluted to 100 μg/mL in 5% ethanol daily and kept at 4°C. The DMPGE$_2$ was administered to rats subcutaneously or intraperitoneally in doses ranging from 50 to 150 μg/kg/day twice daily. Because DMPGE$_2$ appears to be more toxic in the perioperative period, it was not begun until 24 hours after transplantation to allow the animals to fully recover from the surgical stress. Animals received the drug until rejection occurred or for 2 weeks. Control animals underwent an identical procedure and received identical postoperative care, but were given diluent alone.

Mixed Lymphocyte Culture

Mixed lymphocyte cultures were performed in 96-well, flat-bottom microtiter plates using splenic lymphocytes obtained from naive (LBN), recipient (LBN), donor (ACI), and third-party (WF) animals. Spleens were first "teased" apart in a sterile Petri dish containing Hanks' solution. Cell suspensions were then filtered through fine mesh, and red blood cells were lysed with ACK lysing buffer (B+B/Scott Laboratories). Responder lymphocytes were washed three times and were resuspended in culture media (Table 1) at a concentration of 4 × 10^6 cells/mL. Stimulator splenocytes were washed and then irradiated with 3,000 rads from a cesium source and resuspended in media at a concentration of 8 × 10^6 cells/mL. Individual culture wells were plated with 100 μL each of stimulator and responder cell suspensions and were incubated for six days at 37°C in an atmosphere containing 8% carbon dioxide. Six to ten hours before termination, the cells were pulsed with tritiated thymidine. Plates were harvested using the Betaplate harvesting system.

Table 1. Contents of Culture Media (Each 100 mL)

Constituent	Strength (mL)
M-199 with salts[a]	96.00
Rat serum (responder)	2.50
HEPES buffer (1 mol/L)	1.00
2-Mercaptoethanol (0.01 mol/L)	0.10
Penicillin/streptomycin solution (100 U/mL, 100 μg/mL)	0.50

[a] Grand Island Biological Co, Grand Island, NY.

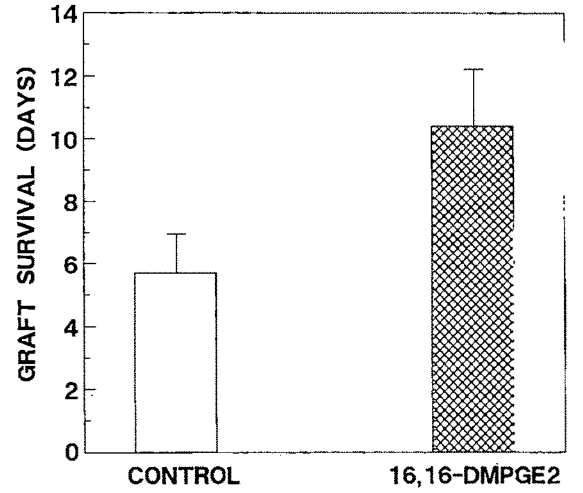
Lymphocytes from the ACI/LBN pair were incubated in concentrations of PGE$_2$ from 10^{-4} to 10^{-9} mol/L using 4 rats from each group per panel of cultures. Additional cultures were performed using splenocytes from transplant recipients, both the control and PGE$_2$-treated groups (no PGE$_2$ was added to these cultures). In this set of experiments, LBN rats (n = 8) received transplants from ACI donors. The animals were treated with either DMPGE$_2$ at 150 μg/kg/day or diluent. Four days after transplantation in the control group and six days after transplantation in the treated group, the spleens were harvested for lymphocytes. Stimulators from donor (ACI) and third-party (WF) rats were used to assess alloreactivity. Naive rats (LBN without a transplant) served as the control group for these experiments.

Results

Rats treated with 25 to 100 μg/kg/day of DMPGE$_2$ had no prolongation of transplant survival. With doses of 200 μg/kg/day or higher, severe diarrhea occurred, and most rats died after two to three days of treatment, a finding confirmed by others to be due to inhibition of small-bowel absorption. Postmortem examination of these animals showed no gross pathological explanation for their death.

Overexposure to DMPGE$_2$ has been shown to cause intense bronchospasm and gastrointestinal disturbances. Thus, death due to dehydration would not seem unreasonable. We were unable to assess the presence of bronchospasm in these animals, but bronchospasm resulting from DMPGE$_2$ compounding the effects of ether anesthesia could explain the observation that even moderate doses (100 to 150 μg/kg/day) given in the immediate perioperative period usually resulted in the death of the animal. No toxicity other than mild diarrhea was seen at a dose of 150 μg/kg/day. Thus, to give the maximum dose of drug not associated with toxicity, we gave the transplanted rats in the treatment group 150 μg/kg/day of DMPGE$_2$ beginning on the day after transplantation.

Survival of grafts treated with 150 μg/kg/day of DMPGE$_2$ (n = 9) was significantly prolonged compared with survival of control grafts (n = 7): 10.4 ± 1.7 days versus 5.7 ± 1.1 days (mean ± standard error of the mean) (p = 0.001) (Fig 1). One animal had a functioning graft more than 100 days after transplantation and was not included in the statistical analysis. There were no differences in complete blood counts, electrolyte levels, serum protein levels, or liver function tests between treatment and control groups. Comparison of transplanted hearts from treated and control groups demonstrated similar patterns of cellular infiltrate, edema, and myocyte necrosis, although these manifestations of rejec-



GROUP	N	SURVIVAL (DAYS)	MEAN ± SEM
CONTROL	7	5, 5, 5, 5, 6, 6, 8	5.7 ± 1.1
DMPGE2	9	10, 10, 13, 9, 9, 12, 8, 12, >100*	10.4 ± 1.7

Fig 1. Allograft survival expressed as days of palpable systolic function for rats treated with 16,16-dimethyl prostaglandin E_2 *(*DMPGE2*) versus control animals. The single graft that achieved indefinite graft survival was not included in the statistical analysis (***). Treatment with* $DMPGE_2$ *nearly doubled survival time. Data are shown as the mean ± the standard error of the mean (*SEM*).*

tion were significantly delayed in the group receiving $DMPGE_2$.

When $DMPGE_2$ was added to mixed lymphocyte cultures using splenocytes from naive LBN and ACI rats, a dose-dependent suppression of the mixed lymphocyte response (MLR) was repeatedly demonstrated. Suppression of the MLR was detectable at $DMPGE_2$ concentrations of 1×10^{-7} mol/L. When the $DMPGE_2$ concentration reached 1×10^{-6} mol/L, suppression was greater than 80% (Fig 2). Splenocytes harvested from treated animals with functioning but histologically rejecting hearts demonstrated a marked decrease in MLR to donor (ACI) stimulators compared with identically stimulated naive LBN controls (3,804 ± 603 cpm versus 27,395 ± 2,668 cpm, n = 4), but showed no suppression of response to third-party (WF) stimulators (10,764 ± 1,230 cpm versus 14,157 ± 1,138 cpm, n = 4) compared with naive responders (Fig 3). Splenocytes harvested from control animals with functioning but histologically rejecting hearts showed decreased responsiveness both to donor (ACI) stimulators and third-party (WF) stimulators compared with naive controls (data not shown).

Comment

The interest in immunosuppression after burn and trauma injury has prompted investigation into the relationship between PGE_2 and IL-2. It is clear that PGE_2 inhibits both the production and the subsequent effects of IL-2 in several models [6, 8, 9]. Because endogenous prostaglandins are rapidly metabolized, either locally or in a single pass through the pulmonary circulation, a synthetic prostaglandin ($DMPGE_2$) was used for the present study. The substitution of two methyl groups at the 16 position blocks the naturally occurring enzymatic degradation of the 15 hydroxyl group and allows a half-life of several hours [10].

Administration of $DMPGE_2$ to heterotopic cardiac allograft recipients nearly doubled graft survival time in this study. Although inhibition of IL-2 activity may play a role in the prolongation of survival, PGE_2 has also been shown to inhibit coagulation, increase blood flow to ischemic areas, and increase intracellular concentrations of cyclic adenosine monophosphate, all actions that could be ex-

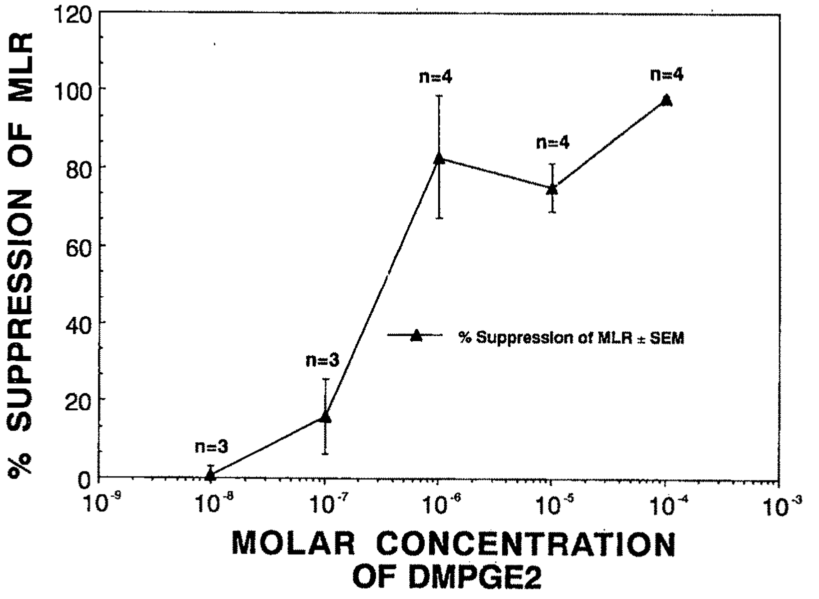


Fig 2. Suppression of $[^3H]$*thymidine incorporation in ACI-stimulated LBN mixed lymphocyte cultures caused by increasing concentration of 16,16-dimethyl prostaglandin* E_2 *(*DMPGE2*). Data are shown as the mean ± the standard error of the mean (*SEM*) of three or four experiments each using the splenocytes from several rats. A dose-dependent suppression of blastogenesis is shown with concentrations as low as* 10^{-7} *mol/L. Percent suppression was calculated as follows:* $1 - \{[cpm(exp) - cpm(self)]/[cpm(allo) - cpm(self)]\} \times 100\%$*, where cpm = counts per minute, exp = experimental wells, and allo = allogeneic. (*MLR *= mixed lymphocyte response; n = number of experiments.)*

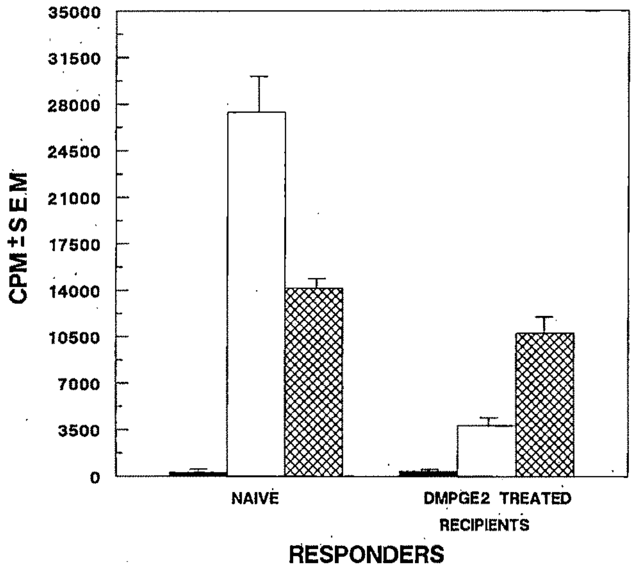


Fig 3. Results of the mixed lymphocyte response of 16,16-dimethyl prostaglandin E_2 (DMPGE2)–treated transplant-recipient (LBN) splenocytes compared with the response of naive LBN splenocytes. Stimulators were irradiated self (LBN, solid bars*), donor (ACI,* open bars*), and third-party (WF,* hatched bars*) splenocytes. Marked suppression of antidonor response (3,804 ± 603 versus 27,395 ± 2,668 cpm) but not anti–third-party response (10,764 ± 1,230 versus 14,157 ± 1,138 cpm) was seen in DMPGE$_2$-treated transplant recipients compared with controls. Data are shown as the mean ± the standard error of the mean* (SEM) *(n = 4).*

pected to enhance allograft survival [10, 11]. However, despite continuous administration of the compound, all but 1 animal eventually rejected its allograft. Further work using DMPGE$_2$ administered continuously to the arterial supply of the graft is planned. This will attempt to exploit the pronounced effects of even minute quantities of DMPGE$_2$ on the local environment while avoiding the toxic manifestations of systemic dosing. Furthermore, studies [12] of graft modification before transplantation have shown that pretreatment of a cardiac graft by perfusion with DMPGE$_2$ affords protection from immunological injury.

Other investigators [13] have shown the dose-dependent suppression of lymphocyte proliferation with administration of PGE$_2$ as demonstrated in the present study. However, we thought it was important to reproduce this effect in the same allogeneic combination used for transplantation. Initially, great difficulty was encountered in obtaining consistent mixed lymphocyte cultures of rat splenocytes. Multiple concentrations of heat-inactivated recipient serum with various stimulator to responder ratios and durations of incubation were tried before reproducible results were achieved using the combinations reported. The decrease in MLR to donor stimulation seen in lymphocytes taken from treated animals was reproducible in all four experiments. Because the response to third-party stimulation was normal, we speculated that the inhibition to donor stimulation may be due to sequestration of alloreactive clones in the graft. However, this would not explain the depression of MLR response both to donor and third-party stimulation seen in untreated allograft recipients (data not shown).

These data have demonstrated that rejection of a rat cardiac allograft is suppressed by DMPGE$_2$, allogeneic MLR is inhibited by DMPGE$_2$ in a dose-dependent manner, and transplanted animals under treatment with DMPGE$_2$ exhibit donor-specific in vitro hyporesponsiveness. The specific mechanism by which DMPGE$_2$ exerts its in vivo and in vitro immunosuppressive action remains unclear and open to speculation at this time.

We thank Dr David Sachs for many helpful discussions about transplantation and for the use of his laboratory for our in vitro experiments.

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DISCUSSION

DR J. KENT TRINKLE (San Antonio, TX): I have two questions. First, does PGE_1 have this effect on IL-2 secretion? I ask because we have all been using it for a long time, and I have never heard that before.

DR ROSENGARD: Prostaglandin E_1 does not have the same immunosuppressive activity as PGE_2. If you study a wide variety of prostaglandins, each one has a different profile in terms of its ability to inhibit alloreactivity, and I do not believe PGE_1 has ever been documented to have this type of immunosuppressive effect.

DR TRINKLE: Thank you. Second, when you get this initial suppression, is there any evidence that it is like OKT3, where if you suppress the reactivity early, the recipient has a depressed ability to develop memory T and B cells and recognize that as a foreign allograft?

DR ROSENGARD: The reason I specifically focused on the 1 animal that survived more than 100 days addresses that point exactly. The fact that you get a profile of reduction of MLR to donor type but not to third-party type, which is induced by the drug in treated animals, would indicate that there is, in fact, that possibility and that perhaps if a larger number of animals were studied, this kind of phenomenon could be detected with much greater frequency. For example, in similar rat cardiac allografts, short courses of treatment with cyclosporine can induce long-term tolerance of the graft, and similar patterns are seen in suppression of donor versus third-party MLR.

Superior Myocardial Preservation With Modified UW Solution After Prolonged Ischemia in the Rat Heart

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Cardiac transplantation remains constrained by poor graft tolerance of prolonged cold ischemia. University of Wisconsin solution has remarkably extended ischemic preservation in pancreas, kidney, and liver transplantation. To assess its efficacy in cardiac preservation, modified University of Wisconsin solution flush and storage were tested against St. Thomas' cardioplegia flush and normal saline solution storage after six hours of ischemia at 0°C in 46 isolated rat hearts. After ischemia, groups were compared before and after reperfusion. After ischemia but before reperfusion, University of Wisconsin solution hearts had significantly less tissue water (3.8%), superior tissue sodium, potassium, calcium, and magnesium profiles, and elevated adenosine and inosine levels, and tended toward better histological preservation. After reperfusion, University of Wisconsin solution more effectively preserved left ventricular compliance (75% versus 35% of baseline), developed pressure (71% versus 45% of baseline), histological integrity, and tissue potassium and calcium profiles than St. Thomas' solution. The University of Wisconsin solution provided superior preservation of systolic and diastolic ventricular function, tissue histology, tissue water, and tissue electrolytes than did St. Thomas' cardioplegia and normal saline solution storage in this experimental model, and might result in improved graft tolerance of ischemia in clinical cardiac transplantation.

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A major remaining problem in cardiac transplantation is poor graft tolerance of prolonged cold ischemia. Acceptable periods of ischemia in clinical practice are currently limited to three to four hours [1, 2]. In laboratory models, longer successful preservation has been demonstrated [3], but these experimental regimens are probably on the verge of clinical safety. Several logistical problems arise as a result of this narrow margin of safety. The potential donor pool is limited by distances involved. Precise coordination is required between the donor and recipient surgical teams. Chartered air transportation is generally required for expedient organ transport, and procedures are often performed as late-night emergencies with the concomitant risk of emergent procedures.

Various myocardial preservation regimens have been employed. Excellent clinical results have been reported with rinse and storage in simple saline solution [1], and success has also been achieved with cardioplegia flush and crystalloid storage [2]. Hypothermic pulsatile perfusion is reported to be more effective for long-term preservation (24 to 48 hours); however, this method not only necessitates complex equipment but might increase the risk of infection [4]. A more effective flush and storage preservation solution might extend allowable periods of cold ischemia as effectively and more conveniently.

Recently, several laboratory and clinical investigators reported remarkably extended cold preservation of pancreas [5], kidney [6], and liver [7] grafts with UW solution, so named because of its conception at the University of Wisconsin as a flush and storage medium for pancreas transplantation. The solution offers potential resolution of many theoretical and practical concerns in organ preservation [8].

This study was designed to test the efficacy of UW solution as a flush and storage solution for extended myocardial preservation. St. Thomas' Hospital cardioplegia (ST) flush with normal saline solution storage was chosen as control to mimic current clinical regimens. Variables of ventricular function, histopathology, tissue water, tissue electrolytes, and adenine nucleotide pool metabolites were employed to assess prolonged myocardial preservation.

Material and Methods

Experimental Protocol and Ventricular Function Assessment

All experimental animals received humane care in compliance with the "Principles of Laboratory Animal Care" (National Society for Medical Research) and the "Guide for the Care and Use of Laboratory Animals" (National Institutes of Health publication No. 80-23, revised 1978).

The experimental protocol is summarized in Figure 1. After baseline metabolite (adenine nucleotide pool) and

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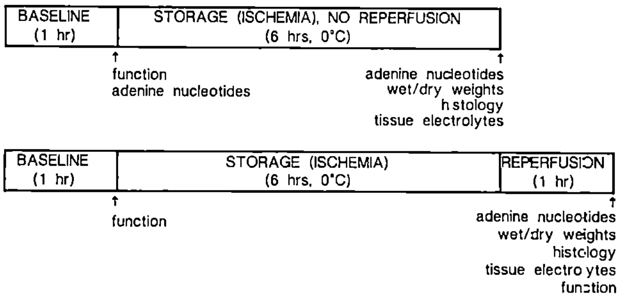

Fig 1. Experimental protocol. Two groups of hearts were tested with each experimental regimen, one after ischemia only and the second after ischemia and reperfusion.

Table 2. Chemical Analysis of the Two Solutions

Component	University of Wisconsin Solution	St. Thomas' Hospital Cardioplegia
Sodium (mmol/L)	30	133
Potassium (mmol/L)	113	16
Calcium (mmol/L)	0.10	1.39
Magnesium (mmol/L)	4.81	15.22
Glucose (mmol/L)	0.06	0.0
Osmolarity (mosm/kg)	323	326
Adenosine (mmol/L)	2.41	0.0
Inosine (mmol/L)	0.00429	0.0
pH	7.7	7.8

mechanical function assessments, isolated hearts were stored for six hours at 0°C with one of two regimens: UW solution flush and storage constituted the experimental regimen, and ST solution flush and normal saline solution storage constituted the control regimen. Complete solution compositions are listed in Table 1. Both were analyzed for chemical content and adenine nucleotide pool metabolites (Table 2). Because of the potential for calcium paradox with the relatively hypocalcemic UW solution [9], calcium (0.5 mmol) was added to each liter of UW solution.

Table 1. Composition of Solutions

Component	Amount
Modified University of Wisconsin Preservation Solution	
Impermeant agents	
Raffinose (mmol/L)	30
Potassium lactobionate (mmol/L)	100
H^+ buffer	
KH_2PO_4 (mmol/L)	25
Colloid	
Hydroxyethyl starch (g/dL)	5
$MgSO_4$ (mmol/L)	5
Adenosine (mmol/L)	5
Glutathione (mmol/L)	3
Allopurinol (mmol/L)	1
Dexamethasone (mg/L)	16
$CaCl_2$ (mmol/L)	0.5
St. Thomas' Hospital Cardioplegia	
NaCl (mmol/L)	110
KCl (mmol/L)	16
$MgCl_2$ (mmol/L)	16
$CaCl_2$ (mmol/L)	1.2
$NaHCO_3$ (mmol/L)	10
Krebs-Henseleit Buffer	
NaCl (g/L)	6.92
$NaHCO_3$ (g/L)	2.10
Dextrose (g/L)	1.98
KCl (g/L)	0.35
$MgSO_4$ (g/L)	0.15
KH_2PO_4 (g/L)	0.16

Two groups of hearts were tested with each regimen. After ischemia (storage), one group of hearts was tested before reperfusion by assessing adenine nucleotide pool metabolites, wet and dry weights, tissue histology, and tissue electrolytes. A second group of hearts was tested after reperfusion. Factors assessed included mechanical function, adenine nucleotide metabolites, wet and dry weights, tissue histology, and tissue electrolytes.

Sprague-Dawley rats (Charles River, Wilmington, MA) weighing approximately 300 g were anesthetized with intraperitoneal administration of sodium pentobarbital, 20 mg/kg, and intravenously heparinized (400 units/kg) through a femoral vein cutdown. Rapid excision of the heart was performed and isolated perfusion initiated for assessment of baseline ventricular function.

An isolated heart column (Langendorff preparation) was employed to perfuse hearts with filtered (10 μm) Krebs-Henseleit buffer (see Table 1) at a constant pressure of 100 cm H_2O. System temperature was maintained at 37°C by jacketing all reservoirs and perfusate conduits with temperature-controlled recirculating water. Krebs-Henseleit buffer was oxygenated and the pH controlled by bubbling with a mixture of 95% oxygen and 5% carbon dioxide. Hearts were secured on an aortic cannula (14-gauge Jelco) that could be rapidly detached to perform sequential studies. All hearts were paced with an S44 Grass stimulator (Grass Instruments, Quincy, MA) and bipolar right atrial leads at 300 beats per minute, 5-ms duration, and the lowest voltage that captured the heart (generally 1 to 2 V, though higher voltages were sometimes required during reperfusion). An intraventricular balloon filled with saline solution was inserted through a left atriotomy, across the mitral valve, and into the left ventricle for isovolumic pressure measurements. Balloon pressures were recorded with a model 1280 pressure transducer, 8805C pressure channel, 8814A derivative computer, and 7758A chart recorder (all: Hewlett-Packard, Atlanta, GA). All measurements were recorded after ventricular stabilization (15 minutes). Total perfusion time required was approximately one hour to generate three sets of functional measurements, the first of which was discarded because of small balloon shifts.

Functional variables assessed were developed pressure, peak positive rate of rise of left ventricular pressure (dP/dt), end-diastolic pressure, and peak negative dP/dt. To assess systolic function at baseline (before storage), these variables were tested at the balloon volume required to give an end-diastolic pressure of 5 mm Hg. After storage, this volume was retested provided end-diastolic pressure remained less than 30 mm Hg. If not, then functional variables were recorded at the highest balloon volume that yielded an end-diastolic pressure of 30 mm Hg, as higher end-diastolic pressures are nonphysiological and of uncertain meaning.

To assess diastolic function, balloon volume was varied in 0.02-mL increments to produce end-diastolic pressures ranging from 0 to 30 mm Hg. Compliance curves were generated by plotting end-diastolic pressure against volume. Because curves were linear in the range of volumes tested, simple linear regressions and x-intercepts were used to compare ventricular compliance between groups.

After baseline functional assessment, hearts were detached from the column and immediately flushed with 10 mL of flush solution for one minute (UW or ST) and then submerged in storage solution (UW or normal saline) for six hours at 0°C. After ischemia, hearts were tested in two groups: (1) after ischemia (storage) only (UW: $n = 12$; ST: $n = 16$) and (2) after ischemia and reperfusion (UW: $n = 9$; ST: $n = 9$).

Baseline adenine nucleotide pool metabolites were assessed immediately after flushing but before storage. Excisional biopsy specimens (10 mg) were taken of full-thickness left ventricular free wall and immediately frozen in liquid nitrogen. After storage with the experimental or control regimen, repeat biopsy was performed. The apex of the heart (approximately 0.5 g of tissue) was analyzed for tissue water and tissue electrolytes. The base of the heart was histologically examined after fixation by flushing with 10 mL of 10% buffered formalin and submersion in the same solution.

Hearts in the reperfusion group underwent six hours of cold storage followed by repeat functional measurements during reperfusion on the isolated heart column. After reperfusion, biopsy specimens of the hearts were obtained for measurement of adenine nucleotide pool metabolites, tissue water determination, and histological examination.

Tissue Water Assessment

Tissue water was determined after storage (in the no-reperfusion group) and after storage and reperfusion (in the reperfusion group) using the apex of the heart. Wet weights were measured after excess fluid was blotted from specimens. Dry weights were measured after the specimens were dried for 24 hours at 70°C. Percent tissue water was determined with the following formula: (wet weight−dry weight)/wet weight.

Histological Assessment

All specimens were fixed in 10% buffered formalin and stained with hematoxylin and eosin. Histological grades ranged from 0 through 4. The grade represented a composite of number of damaged myocytes and number of foci of damage. Damaged myocytes were defined as cells with any combination of waviness, contraction bands, disruption of plasma membranes, or loss of myofibril definition. The number of foci was determined in a single cross-section of myocardium taken at the specimen's widest diameter. The grades were as follows: grade 0, no evidence of ischemic injury; grade 1, had one to five foci of one to three damaged myocytes per focus; grade 2, six to ten foci of one to three damaged myocytes per focus or one to five foci of four to six damaged myocytes per focus; grade 3, more than ten foci of one to three damaged myocytes per focus or six to ten foci of four to six damaged myocytes per focus; and grade 4, any number of foci of damaged myocytes with more than six damaged myocytes per focus. All specimens were examined by a cardiac pathologist blinded to group identification.

Adenine Nucleotide Pool Metabolite Assessment

Adenine nucleotide pool metabolites were extracted from the biopsy specimen as previously described [10]. With temperature maintained at 4°C throughout the extraction process, tissue was homogenized in 0.4 mL of 12% trichloroacetic acid for 30 minutes. The soluble extract was separated from denatured protein by centrifugation at 2,000 *g* for ten minutes. The supernatant was neutralized with 0.8 mL of tri-*N*-octylamine and Freon (1:3 vol/vol) and vortexed for one minute. This mixture was centrifuged at 2,000 *g* for two minutes and the supernatant frozen at −70°C for later high-performance liquid chromatography elution.

Nucleotide elutions were performed using reversed-phase high-performance liquid chromatography as previously described [11]. The following were identified in the myocardial extracts: adenosine triphosphate (ATP), adenosine diphosphate, adenosine monophosphate, xanthine, hypoxanthine, adenosine, inosine, and nicotinamide adenine dinucleotide. All values were standardized to the amount of protein in the original biopsy specimen [12] and expressed as nanomoles per milligram of protein.

Atomic Absorption

All glassware was acid washed in a 1:1 mixture of concentrated sulfuric and nitric acid and then rinsed with distilled, deionized water. A Video 22 atomic absorption spectrophotometer (Thermo Jarrell Ash Corporation, Franklin, MA) was used to determine metal ion content. Standard curves were prepared with dilutions of certified reference solutions (Fisher Scientific, Springfield, NJ). All samples and standards were prepared with 5% nitric acid in distilled, deionized water. Samples were ashed in quartz crucibles in a muffle furnace for eight hours at 550° to 650°C. The ash was digested for 30 minutes with 0.25 mL of concentrated nitric acid and then diluted with distilled, deionized water to a total volume of 5 mL and calcium determinations were performed. Of this solution, 0.5 mL was diluted to 50 mL, and sodium, potassium, and magnesium determinations were performed. Three atomic absorption spectrophotometric readings were taken for each sample and averaged to give a final concentration (μg/mL). These results were expressed as

Table 3. Ventricular Function[a]

Variable	Developed Pressure (mm Hg)	Peak +dP/dt (mm Hg/s)	Slope (mm Hg/mL)	X-Intercept (mL)	Shift X-Intercept (mL)	Peak −dP/dt (mm Hg/s)
Baseline (Before Storage)						
UW	143 ± 5	4,594 ± 237	203 ± 19	0.28 ± 0.03	. . .	−2,740 ± 129
ST	150 ± 8	4,364 ± 272	212 ± 27	0.31 ± 0.02	. . .	−2,747 ± 153
p Value	0.4890	0.5322	0.7864	0.4175	. . .	0.9739
Ischemia and Reperfusion						
UW	101 ± 7	3,317 ± 140	218 ± 11	0.21 ± 0.04	−0.07 ± 0.04	−2,193 ± 155
ST	68 ± 8	1,850 ± 184	1,083 ± 458	0.11 ± 0.03	−0.20 ± 0.04	−1,347 ± 131
p Value	0.0047[b]	0.0000[b]	0.0770	0.0785	0.0292[b]	0.0007[b]

[a] Data are shown as the mean ± the standard error of the mean. [b] Differences are significant ($p < 0.05$).

+dP/dt = peak positive rate of rise of left ventricular pressure; −dP/dt = peak negative rate of rise of left ventricular pressure; ST = St. Thomas' Hospital Cardioplegia; UW = University of Wisconsin solution.

micrograms of electrolyte per gram of dry myocardial tissue.

Statistical Analysis

Statistical analysis was performed using SAS statistical analysis systems (SAS Institute Inc, Cary, NC). Unpaired *t* tests were used for comparisons between experimental and control groups.

Results

Ventricular Function

Baseline functional data (before ischemia) showed no significant differences between groups (Table 3). Preservation of systolic function was significantly better with UW solution. At baseline, UW hearts had a mean developed pressure of 143 mm Hg versus 150 mm Hg for ST hearts. After storage, mean developed pressure of UW hearts was 101 mm Hg (71% of baseline) versus 68 mm Hg (45% of baseline) for the ST hearts. Mean peak positive dP/dt was improved in UW hearts versus ST hearts: 3,317 mm Hg/s (72% of baseline) versus 1,850 mm Hg/s (42% of baseline).

Postischemic diastolic function revealed significantly better compliance with UW solution than ST solution (Fig 2). Loss of compliance was assessed by measuring the shift of the x-intercept of compliance curves generated before and after storage. At baseline, UW hearts had a mean x-intercept of 0.28 mL and ST hearts, 0.31 mL. After storage and reperfusion, ST hearts shifted the x-intercept of their compliance curves to the left significantly more than UW hearts: 0.20 mL (75% of baseline) versus 0.07 mL (35% of baseline), respectively. Peak negative dP/dt was significantly higher with UW solution than ST solution: −2,193 mm Hg/s (80% of baseline) versus −1,347 mm Hg/s (49% of baseline). The average slope of compliance curves generated in UW hearts was 218 mm Hg/mL versus 1,083 mm Hg/mL in ST hearts, a difference that approached significance ($p = 0.077$).

Several interesting subjective observations were made in the course of the experiment. Hearts arrested more quickly with UW solution (one to two beats) than ST solution (five to six beats). When hearts were handled after storage, UW hearts retained their prestorage compliant consistency, but ST hearts were firm and rubbery. Virtually all UW hearts spontaneously defibrillated within seconds of reperfusion, whereas ST hearts fibrillated for several minutes and often required mechanical defibrillation before regaining organized rhythm. The ST hearts slowly regained limited systolic function (developed pressure and peak positive dP/dt) over one hour of reperfusion. The UW hearts initially functioned at a high percentage of baseline but lost a percentage of this function during the first few minutes of reperfusion. In spite of this loss, UW hearts stabilized with developed pressures and peak positive dP/dts significantly higher than ST hearts, as already discussed.

Tissue Water, Tissue Electrolytes, and Histology

Percent tissue water before reperfusion was significantly lower in hearts preserved with UW solution versus ST solution: 75.91% versus 79.71% (Table 4). This difference disappeared with reperfusion.

All tested tissue electrolytes were significantly different between groups before reperfusion (Fig 3). Compared with ST hearts, UW hearts had lower sodium contents

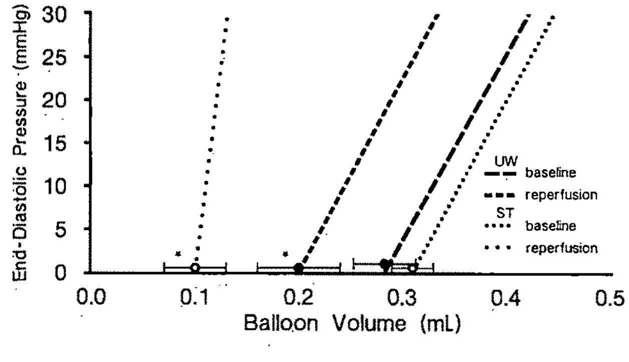

Fig 2. Mean ventricular compliance (end-diastolic pressure versus balloon volume). There is a leftward shift, ie, shift in x-intercept of diastolic pressure–volume relationship, of St. Thomas' Hospital cardioplegia (ST) *hearts versus University of Wisconsin solution* (UW) *hearts at baseline and with reperfusion.* (* = $p < 0.05$.)

Table 4. Tissue Water and Histology[a]

Variable	Tissue Water (%)	Histological Grade
Ischemia Only		
UW	75.91 ± 0.21	1.28 ± 0.16
ST	79.71 ± 0.12	1.69 ± 0.12
p Value	0.0000[b]	0.0534
Ischemia and Reperfusion		
UW	81.52 ± 0.37	2.25 ± 0.18
ST	82.15 ± 0.48	2.90 ± 0.18
p Value	0.3035	0.0195[b]

[a] Data are shown as the mean ± the standard error of the mean. [b] Differences are significant ($p < 0.05$).

ST = St. Thomas' Hospital Cardioplegia; UW = University of Wisconsin solution.

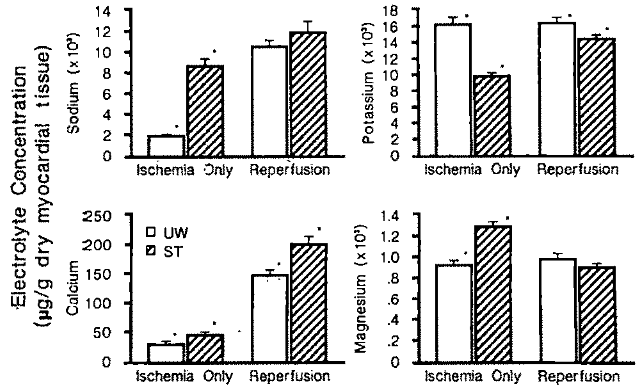

Fig 3. Tissue electrolytes of University of Wisconsin solution (UW) *hearts versus St.Thomas' Hospital cardioplegia* (ST) *hearts after six hours of storage before and after reperfusion.* (* = p < *0.05.)*

(2,014 versus 8,774 µg/g; $p = 0.0001$), higher potassium contents (16,245 versus 9,904 µg/g; $p = 0.0001$), lower calcium contents (31.4 versus 46.9 µg/g; $p = 0.0044$), and lower magnesium contents (932 versus 1,299 µg/g; $p = 0.0001$). After reperfusion, significant differences persisted between UW heart and ST heart potassium contents (16,473 versus 14,503 µg/g; $p = 0.0214$) and calcium contents (149 versus 202 µg/g; $p = 0.0010$) (see Fig 3). Differences between tissue sodium content (10,631 versus 11,967 µg/g; $p = 0.2190$) and magnesium content (987 versus 908 µg/g; $p = 0.1615$) were no longer significant.

Before reperfusion, blinded histological evaluation approached significance with a mean histological grade of 1.28 for ST hearts versus 1.69 for UW hearts ($p = 0.053$). After reperfusion, significantly better histological grades were noted for hearts preserved with UW (2.25) versus ST solution (2.90).

Adenine Nucleotide Pool Metabolites

Before ischemia (storage), UW hearts demonstrated higher levels of adenosine (7.82 versus 0.10 nmol/mg protein) and higher levels of inosine (1.06 versus 0.15 nmol/mg protein) (Table 5). These differences persisted after storage but before reperfusion. Adenosine levels remained elevated at 8.62 versus 0.88 nmol/mg protein, but were not significantly changed from baseline. Inosine levels not only remained elevated but increased significantly from baseline to 7.37 (UW) versus 3.34 (ST) nmol/mg protein. Hypoxanthine levels were also significantly elevated in UW versus ST hearts after storage (1.83 versus 1.30 nmol/mg protein), though no differences were noted before storage. All differences in adenine nucleotide metabolites between UW and ST groups disappeared after reperfusion.

Comment

Myocardial preservation using UW solution flush and storage was tested against ST solution flush with normal saline solution storage in an isolated rat heart model. The ST solution is a commonly used standard extracellular cardioplegia formulation [8]. The formulation of UW solution addresses several theoretical and practical concerns: (1) minimizing hypothermia-induced cell swelling, (2) preventing intracellular acidosis, (3) preventing interstitial space expansion during the flush-out period, (4) preventing oxygen free radical–mediated injury during reperfusion, (5) providing substrates for high-energy

Table 5. Adenine Nucleotide Metabolites[a]

Group	Adenosine Triphosphate	Adenosine Diphosphate	Adenosine Monophosphate	Adenosine	Inosine	Hypoxanthine	Xanthine
Baseline at Onset of Ischemia, Before Storage							
UW	14.46 ± 1.10	7.77 ± 0.23	3.98 ± 0.46	7.82 ± 0.57	1.06 ± 0.28	0.83 + 0.15	4.56 ± 0.26
ST	14.95 ± 0.75	8.42 ± 0.26	3.55 ± 0.31	0.10 ± 0.05[b]	0.15 ± 0.07[b]	0.74 + 0.19	4.55 ± 0.89
Ischemia Only							
UW	3.30 ± 0.70	3.87 ± 0.44	8.94 ± 0.91	8.62 ± 1.18	7.37 ± 0.64	1.83 ± 0.16	3.35 ± 0.29
ST	2.73 ± 0.59	3.82 ± 0.42	8.45 ± 0.57	0.88 ± 0.17[b]	3.34 ± 0.35[b]	1.30 ± 0.07[b]	2.87 ± 0.12
Ischemia and Reperfusion							
UW	8.94 ± 0.85	5.79 ± 0.28	5.12 ± 0.65	0.61 ± 0.11	0.57 ± 0.11	0.75 + 0.12	3.52 ± 0.14
ST	8.35 ± 1.23	5.13 ± 0.59	3.97 ± 0.32	0.64 ± 0.12	0.46 ± 0.14	0.60 + 0.11	3.64 ± 0.41

[a] Data are shown as the mean ± the standard error of the mean in nanomoles per milligram of protein. [b] Significance: $p < 0.05$ between the two groups.

ST = St. Thomas' Hospital cardioplegia; UW = University of Wisconsin solution.

phosphate repletion during reperfusion, and (6) maintaining a homeostatic intracellular environment [7].

To date, hypothermia remains the most important and effective factor in organ preservation [13], largely as a result of reduced tissue metabolism. However, the benefits of hypothermia are partially offset by hypothermia-induced cell swelling [14]. Normally cells are bathed in extracellular fluid high in sodium and low in potassium concentration relative to intracellular concentrations. This gradient is maintained by the plasma membrane's Na-K ATPase, which utilizes much of the high-energy phosphate stores derived from oxidative phosphorylation. Anaerobic hypothermic preservation suppresses activity of Na-K ATPase and decreases the ability of the cell to maintain its normal plasma membrane electrochemical gradient and therefore its volume-regulating capacity [15]. As this activity is suppressed, sodium and chloride enter the cell down their concentration gradients, and cellular swelling occurs as water passively migrates to equalize intracellular and extracellular osmotic pressures. Cellular swelling is accompanied by cellular damage [13, 16]. UW solution contains two nonmetabolized, osmotically active impermeant agents with large molecular weights: potassium lactobionate (358 MW) and the saccharide raffinose (594 MW). They were found by Wahlberg and colleagues [17] in early experiments to be effective impermeant agents in pancreas tissue slices, in theory by suppressing hypothermically induced cell swelling and obviating the damage caused by cell swelling [7].

The rationale for the remaining components of UW solution are as follows: (1) To prevent intracellular acidosis resulting from anaerobic metabolism and glycolysis a hydrogen ion buffer, KH_2PO_4, is added. (2) To prevent expansion of the interstitial space that might compress the capillary system and worsen solute exchange between the interstitial space and the capillary beds, a specially prepared fraction of hydroxyethyl starch is used, which in theory blocks interstitial edema by blocking interstices in the basement membrane [18]. (3) To prevent injury by oxygen free radicals during reperfusion, allopurinol, a xanthine oxidase inhibitor, is added to suppress xanthine oxidase–catalyzed generation of free radicals. (4) Glutathione is added to chemically reduce cytotoxic oxidants and suppress lipid peroxidation as well as to prevent loss of endogenous glutathione from the cell. (5) Adenosine is added to facilitate regeneration of ATP during reperfusion by providing a precursor for its formation. (6) Finally, magnesium is added for its membrane-stabilizing properties.

Swanson and co-workers [19] tested the efficacy of UW solution in myocardial preservation with an isolated working canine heart preparation using storage times of five and 12 hours. Comparisons were made against Stanford and modified Collins's solution. No differences between regimens were noted after five hours of storage; however, after 12 hours of storage, ventricular function, tissue water, and sodium as well as oxygen and lactate utilization were better preserved with UW solution after 150 minutes of reperfusion. Based on the relatively high rate of adenine nucleotide catabolism in ischemic rat myocardium compared with canine myocardium, six hours of ischemia in the rat model may be equivalent to 12 or more hours of ischemia in the canine model [20].

In testing ventricular systolic and diastolic function over a range of balloon volumes, if a given balloon volume produced an end-diastolic pressure greater than 30 mm Hg, higher volumes were not tested so as not to interfere with later histological evaluation of tissue damage. As ST hearts often had reduced compliance after ischemia, maximal developed pressure may not have been manifested at tested balloon volumes. If these volumes had been tested, however, the physiological meaning of developed pressure in the setting of such unphysiological end-diastolic pressures would not have been clear. With such poor ventricular compliance, orthotopic transplantation of these hearts is unlikely to have resulted in animal survival because poor diastolic filling would have precluded effective systolic function. On the basis of diastolic data alone, therefore, ST hearts could be classified as nonviable. Nevertheless, at the balloon volumes tested, preservation of systolic function of UW hearts was significantly better than that of ST hearts.

The subjective observations of more pliable myocardium and more rapid and spontaneous defibrillation of UW-stored hearts correlate with objective data presented. The loss of some early systolic function of UW hearts with reperfusion is a cause of concern, but even with this loss, UW hearts stabilized with significantly better systolic function than ST hearts. The UW solution more quickly arrested the heart, probably a result of its higher potassium concentration.

Before reperfusion, myocardium stored in UW solution had lower tissue water, lower sodium, higher potassium, lower magnesium, and lower calcium contents than myocardium stored in ST solution. The finding of lower tissue water in UW hearts may simply be a passive osmotic phenomenon from the impermeant agents (raffinose and lactobionate) or a blocking of transcapillary fluid transport at the capillary basement membrane level by the hydroxyethyl starch derivative in UW solution [18]. Alternatively, myocytes in these hearts may have been better able to actively maintain their normal sodium potassium gradient by maintaining normal ion transport and hence normal intracellular volumes. Their Na-K ATPases or their membrane integrities may have been better preserved. These data correlate well with later reperfusion and functional testing. The ST hearts had reduced ventricular compliance (an expected finding, as tissue water was elevated), whereas UW hearts had better compliance (and lower tissue water before reperfusion). Not surprisingly, systolic function was also inferior in myocardium whose cells were less able to maintain normal tissue electrolytes. The tendency toward less severe histological injury of UW hearts is also in agreement with these findings.

After reperfusion, differences in tissue water noted before reperfusion disappeared as did differences in adenine nucleotide metabolites between groups (to be discussed). In addition, differences in tissue sodium and magnesium disappeared, although significant differences in tissue potassium and calcium persisted. The equalization of some variables suggests substantial reperfusion injury in both groups; however, the superior mechanical function, superior histological preservation, and superior tissue potassium and calcium profiles afforded by UW solution confirm its superiority, even after reperfusion.

Adenine nucleotide metabolites were examined three times: (1) before ischemia (before storage), (2) after ischemia without reperfusion, and (3) after ischemia with reperfusion. Before ischemia, elevated adenosine and inosine levels may simply represent a combination of solution composition (UW solution contains adenosine) and a clinging of this relatively viscous solution to biopsy specimens. After ischemia, this explanation is less plausible in accounting for inosine elevations because of the time-dependent increase of inosine in tissue specimens while adenosine levels remained relatively stable. It seems more likely that this increase represents metabolism of native and exogenous adenosine to inosine, just as the significantly elevated hypoxanthine level probably represents the metabolism of inosine to hypoxanthine. The original hypothesis that adenosine aids ATP regeneration by serving as substrate is not supported by these data.

The more likely source for ATP regeneration in rodents is probably adenosine monophosphate. Abd-Elfattah and colleagues [20] have shown that the activity of 5′-nucleotidase varies between species and that rodents in particular have less active 5′-nucleotidase. Therefore, rats accumulate adenosine monophosphate rather than adenosine as products of ATP utilization, and it is adenosine monophosphate that serves as the primary substrate for ATP regeneration in rodents. Nevertheless, neither UW or ST solution seemed to provide a more favorable adenine nucleotide pool for recovery. The ultimate fate of adenosine in UW solution is interesting for two reasons. First, adenosine has the potential to be metabolized to inosine and subsequently to hypoxanthine and then xanthine. These adenine nucleotide metabolites can serve as substrates for subsequent free radical formation and contribute to the reperfusion injury already discussed [10]. Second, ATP may be resynthesized from adenosine through the salvage pathway, an energetically faster and less costly way for the cell to replenish its high-energy phosphate store than the de novo pathway of ATP synthesis [21].

Finally, caution is warranted in generalizing these data to humans. Although controversy exists, Downey and associates [22] have demonstrated lower myocardial xanthine oxidase activity in humans than in rats. Therefore, allopurinol, a xanthine oxidase inhibitor, might not be as protective in hearts with low xanthine oxidase activity, possibly diminishing the overall efficacy of UW solution in humans.

Several considerations become important in future solution modification and use. Could the addition of other free radical scavengers more effectively block free radical formation and ameliorate the persistent reperfusion injury previously noted? Alternatively, could metabolic blockade with agents such as adenosine deaminase inhibitors and nucleoside transport blockers trap adenosine in the cell and prevent its breakdown so that it might be more efficiently conserved through the more efficient salvage pathways of ATP synthesis [10]? Use of a more tailored reperfusion solution in subsequent studies will separate further the protective effects of UW solution during ischemia. As a final concern, testing of UW solution using this acute model has not addressed the long-term effect of the solution on the transplanted heart. In vivo transplantation studies with longer-term follow-up will address this question.

Several interesting avenues exist for future investigation. Though not to detract from the apparent effectiveness of UW solution, it represents somewhat of a "shotgun" approach to organ preservation. As discussed, UW contains many compounds directed at practical and theoretical concerns. The role of all these compounds in cardiac preservation is not clear. Which combination of the individual components of the solution is responsible for its demonstrated effects? In liver transplantation, Jamieson and co-authors [23] provided evidence that lactobionate, raffinose, and glutathione are the only compounds essential for the solution's observed effects. They hypothesized that the primary components required for successful cold storage are the appropriate impermeant agents in appropriate concentrations to suppress hypothermically induced cell swelling and that glutathione adds some protection to the solution to reduce oxygen free radical formation. Because working myocardium has a higher rate of oxygen consumption per gram of tissue than liver or kidney grafts [24] and requires more time to fully recover organ function, the findings of Jamieson and colleagues [23] might or might not be directly applicable in myocardial preservation. Furthermore, antioxidants might be more germane during reperfusion than during storage because oxygen free radical damage occurs primarily during reperfusion. Antioxidants might ameliorate this damage if supplied continuously rather than being allowed to wash out during early reperfusion

In conclusion, UW solution flush and storage preserved systolic and diastolic left ventricular function more effectively than ST solution and normal saline solution storage. The UW hearts lost a percentage of their early systolic function within the first few minutes of reperfusion but, even after stabilization, functioned significantly better than hearts stored using the ST regimen. Perhaps solution modification can ameliorate this reperfusion injury and specific component effects can be better delineated in myocardial preservation, but the superior preservation of myocardial function by UW solution and the correlation of this function with superior tissue water, tissue electrolyte, and histological profiles seem quite promising.

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DISCUSSION

DR ROBERT A. GUYTON (Atlanta, GA): This was a very interesting paper. I think it confirms our thoughts that the St. Thomas' solution, the normal extracellular solution that is often used for intraoperative myocardial protection, seems to be a dangerous solution for prolonged cardiac preservation for transplantation compared with solutions containing agents with oncotic activity such as the Stanford solution or the UW solution. I wonder whether you have tested any extracellular solutions, for example, the original Stanford solution, that contain such agents?

DR YEH: No, we have not tested other extracellular solutions, but I think you are correct in targeting the importance of oncotic activity. One of the most promising effects of UW solution is its ability to prevent the pronounced increase in tissue water seen with prolonged storage.

Role of Lung Decortication in Symptomatic Empyemas in Children

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Despite appropriate antibiotics and pleural drainage, the condition of some children with empyema fails to improve. In a 5-year period, 10 children ranging in age from 2 to 16 years underwent lung decortication for a refractory, symptomatic empyema, which had developed 3 to 5 weeks after an initial pneumonic infiltrate. Responsible organisms included β-hemolytic streptococci, *Haemophilus influenzae*, or *Streptococcus pneumoniae* in 6 children. Negative cultures were found in 4 children. The initial computed tomographic scan of the chest in 4 of 8 patients showed more than 75% limitation of lung expansion by the contents of the empyema cavity. In 4 other patients, an extensive pleural peel was seen on initial computed tomographic scan of the chest. Several studies also showed cystic lesions in the collapsed lung. Multiple computed tomographic scans in 3 patients confirmed the lack of clinical and chest roentgenographic improvement with conservative therapy. At decortication in each, the visceral and parietal pleural peel was completely removed, freeing the trapped lung. Two patients also had a concomitant lobectomy for a necrotic right upper lobe (1 patient) and left lower lobe (1). Clinical improvement was marked, with return of temperature, white blood cell count, and appetite to normal. Postoperative morbidity was minimal. Analysis of these patients in whom traditional conservative therapy failed suggested that the initial management during the early exudative phase was often delayed and was not aggressive enough from the standpoint of pleural drainage. Early decortication in these highly select patients with symptomatic, refractory empyemas shortened the hospital stay, minimized the risks of long-term use of antibiotics and prolonged chest tube drainage, completely reexpanded the trapped lung, and led to excellent long-term results.

(Ann Thorac Surg 1990;49:940–7)

Controversy still exists as to the role of lung decortication in the current management of childhood postpneumonic empyema. An empyema in childhood is usually a sequela of bacterial pneumonia, although it can occur after trauma, intrathoracic esophageal perforation, pneumothorax, or thoracic operations. The pathological response to an empyema can be divided into three distinct phases [1]. (1) The immediate response is an *exudative* phase, with outpouring of thin fluid, which has a low cell count and can be removed by thoracentesis. (2) The *fibropurulent* phase is characterized by large quantities of pus, with many white blood cells and fibrin. As the fluid thickens, loculation begins and the lung becomes progressively less expandable. (3) Fibroblasts grow into the exudate on both visceral and parietal pleural surfaces, producing a membranous "peel." With increasing fibrosis, the process becomes chronic and the lung becomes more firmly fixed—the *organizing* phase. Entrapment usually begins within 3 to 4 weeks after the onset of the pleural effusion. Principally because of a better understanding of the pathophysiology of empyemas and the use of newer antibiotics, the mortality rate associated with childhood empyemas has substantially declined to about 1% to 2% since the mid-1970s [2].

The goals of any treatment of an empyema should remain as outlined by Mayo and co-authors [3] in 1982: (1) to save life, (2) to eliminate the empyema, (3) to reexpand the trapped lung, (4) to restore mobility of the chest wall and diaphragm, (5) to return respiratory function to normal, (6) to eliminate morbidity, and (7) to reduce the duration of hospital stay. Although many investigators [4, 5] believe that essentially all patients with childhood empyema can be adequately treated by conventional therapy using chest tube drainage and antibiotics, more recent reports have attempted to identify the role of a minithoracotomy procedure [6] or an open thoracotomy and complete lung decortication in the treatment of postpneumonic empyema [3, 7–9]. Clearly some children do respond poorly or incompletely to conventional therapy.

The timing of presentation, the duration of symptoms, the host resistance, the changing virulence of the infecting organisms, and the initial medical management account for the wide variation in the spectrum of pediatric empyemas, thus complicating the appropriate therapeutic decisions. This study reviews the experience at West Virginia University Hospital from 1983 to 1988 with 10 children who underwent lung decortication for a refractory, symptomatic postpneumonic empyema and emphasizes the treatment decisions that led to early operative intervention. During this time, 31 patients, treated primarily at our

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Table 1. Summary of Preoperative Patient Data

Patient No.	Age (yr)	Bacteria Isolated	No. of Antibiotics	Duration of Symptoms Before Antibiotics (days)	Duration of Local Treatment Before Transfer (days)	Duration of WVU Treatment Before Decortication (days)
1	2	β-Hemolytic streptococci (P, O)	5	13	4	3
2	2	*Streptococcus pneumoniae* (B, P, O)	2	14	1	10
3	2	Negative	5	4	4	5
4	13	Negative	2	21	27	9
5	16	*Haemophilus influenzae* (S)	3	14	10	15
6	2	*Streptococcus pneumoniae* (B, P)	4	14	5	6
7	6	β-Hemolytic streptococci (S)	4	5	5	2
8	3	*Haemophilus influenzae* (P, B)	3	6	4	1
9	8	Negative	2	3	7	3
10	2	Negative	5	10	8	4

B = blood culture; O = operative culture; P = pleural fluid culture; S = sputum culture; WVU = West Virginia University.

institution, responded favorably to conventional therapy and were thus excluded from this study.

Material and Methods

Patient Population

In all 10 patients, the empyema resulted from a pneumonic process. There were 5 boys and 5 girls ranging in age from 2 to 16 years (Table 1). The patients complained of symptoms of a respiratory tract infection with malaise, fever, cough, and some shortness of breath. Occasionally, nausea, vomiting, or chest pain was a dominant complaint. All patients were initially treated locally by the family physician either as an outpatient (4 patients) or as an inpatient (6). The duration of symptoms before antibiotic treatment ranged between three days and 3 weeks. The children were treated with antibiotics for one to 27 days (mean period, six days) before transfer. The patients had usually been treated with at least two different antibiotics without clinical improvement. Only 1 child had a thoracentesis, and none had a chest tube inserted before transfer.

Review of the chest roentgenograms obtained by the referring physician showed a localized pulmonary infiltrate in 5 patients, an infiltrate with a pleural effusion in 2 patients, and complete opacification of one hemithorax in 3 patients (Fig 1). Follow-up chest roentgenograms during local treatment with antibiotics only showed no improvement in the 3 patients with an opacified hemithorax (Fig 2). The pulmonary process in the 5 patients with a localized infiltrate progressed to complete opacification of the hemithorax in 3 patients, and a major pleural effusion developed in the other 2 patients (Figs 3, 4). In the 2 patients with an initial infiltrate and pleural effusion, the pleural effusion enlarged during follow-up. Therefore, on transfer, 6 patients already had a completely opacified hemithorax and 4, an enlarging pleural effusion.

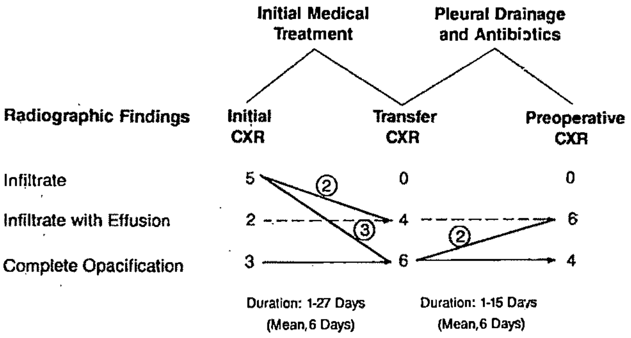

Fig 1. Evaluation of treatment according to findings on chest roentgenograms (CXRs).

Soon after admission, thoracentesis was performed in all patients to obtain pleural fluid for Gram stain and culture. Multiple blood, urine, and sputum cultures were also grown. All children were started on a regimen of two intravenous antibiotics. The antibiotic coverage was subsequently changed dependent on the culture and sensitivity results and the clinical status of the patient. The infecting organism was isolated from the pleural fluid in 4 patients: *Haemophilus influenzae* in 1 patient, β-hemolytic streptococci in 1, and *Streptococcus pneumoniae* in 2 patients. Sputum cultures for β-hemolytic streptococci (1 patient) and *H influenzae* (1) were the only positive isolates in 2 patients. Blood cultures were positive for *H influenzae* (1 patient) and *S pneumoniae* (2) in 3 patients, who also had positive pleural fluid cultures. All cultures were negative in 4 patients, who had been on a regimen on multiple antibiotics at the time of admission.

Closed pleural drainage with a large intercostal tube was established in all patients, as thoracentesis failed to substantially improve the findings on chest roentgenogram. The length of time these patients were treated with antibiotics and closed pleural drainage before proceeding to decortication ranged between one day and 15 days (mean time, six days) (see Table 1, Fig 1). Serial chest roentgenograms continued to show complete opacification of the hemithorax in 4 patients despite chest tube drainage (see Fig 1). In 6 patients (2 with complete hemithorax opacification), there was some improvement

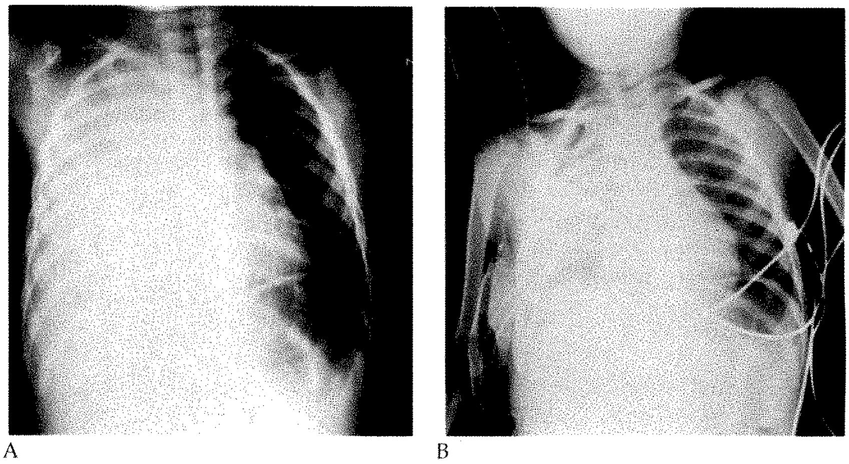

Fig 2. (Patient 6.) (A) Initial chest roentgenogram shows complete opacification of the right hemithorax. After five days of antibiotics, the transfer chest roentgenogram was unchanged. (B) Pleural drainage was established with minimal improvement in the chest roentgenogram. A loculated pneumothorax is noted.

in the chest roentgenogram with pleural drainage. In 3 patients, more than one chest tube was inserted in an effort to facilitate better drainage.

Chest computed tomography was performed in 8 patients to better define the configuration of the pleural fluid collections and the degree of lung compression, and to differentiate parenchymal from pleural involvement. The initial chest computed tomographic (CT) scan in 4 patients showed major (>75%) limitation of lung expansion by loculations of exudate and fluid (Fig 5). A follow-up chest CT scan in 3 of these patients confirmed the lack of both clinical improvement and substantial improvement on the chest roentgenogram with conventional therapy. In 4 patients, the initial chest CT scan revealed an extensive pleural peel entrapping the lung, often with multiple loculations between the collapsed lung and the chest wall that were considered to be no longer amenable to continued pleural drainage and antibiotics (Fig 6). Two scans also showed cystic lesions in the collapsed lung (Fig 7).

The failure of conventional treatment and subsequently the decision to proceed with open thoracotomy and complete lung decortication were based on persistent daily fevers (10 patients), continued elevated white blood cell counts (15,000 to 39,000/μL) (9 patients), a baseline tachycardia (4 patients), tachypnea (8 patients), shortness of breath on activity (5 patients), and signs of serious generalized illness (6 patients). All children had three or more of these findings. Two patients were intubated and on a ventilator because of respiratory distress. Central venous hyperalimentation had been initiated in 2 patients before transfer.

Operative Procedure

The operation was performed under general endotracheal anesthesia using a single-lumen endotracheal tube. A standard posterolateral thoracotomy incision was made, and the pleural space was entered through the fifth intercostal space. Rib resection was not necessary to gain exposure. Invariably, the disease process was worse than expected. The intrapleural gelatinous debris and fibrin mass were evacuated, but there was no inclination for the lung to reexpand without formal decortication (Fig 8). Therefore, the fibrinous peel on the surface of the visceral and parietal pleurae was carefully removed. A plane of cleavage between the visceral pleura and the peel could usually be initially started in the interlobar fissure. As the decortication was usually performed early, the peel was often pliable and stripped away from the lung and chest wall without major air leaks or substantial blood loss. In several patients, the thick peel was adherent, necessitat-

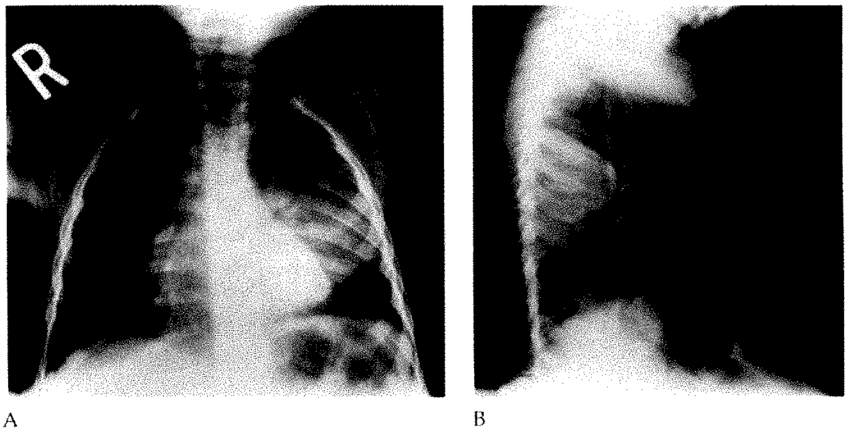

Fig 3. (Patient 10.) (A) Posteroanterior and (B) lateral initial chest roentgenograms show a localized infiltrate in the left lower lobe, for which the patient was treated with antibiotics.

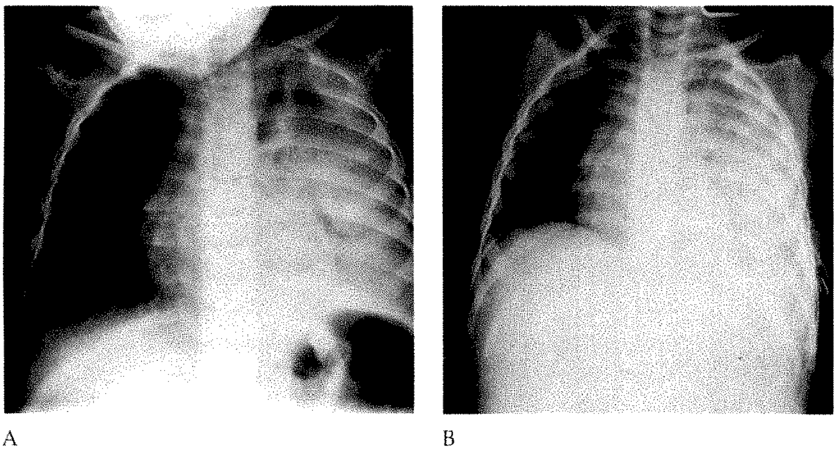

Fig 4. (Patient 10.) (A) Posteroanterior chest roentgenogram demonstrates a major pleural effusion by the time of transfer. (B) With conventional therapy, the patient's clinical status and chest roentgenograms did not improve.

ing tedious dissection and patience to minimize air leaks. Positive-pressure ventilation permitted reexpansion of trapped areas of lung and aided in identifying the cleavage plane. The fibrous peel was also removed from the cardiophrenic and costophrenic sulci and the diaphragm to improve diaphragmatic motion. Lung expansion was immediate and quite gratifying in all patients.

The operative findings were staged according to the three phases of empyema development. Six patients had gelatinous exudate compressing the lung and a pliable visceral peel entrapping the lung (phase II), and in 4 patients an organizing visceral and parietal peel had begun to develop (phase III). Two patients (patients 3 and 6, see Table 1) were found to have extensive necrosis of a lobe of the lung and thus underwent concomitant left lower lobectomy and right upper lobectomy, respectively. The necrotic pulmonary process involved more than 50% of the lobe and was confirmed by subsequent pathological examination. The pleural space was irrigated with an antibiotic solution and drained with two large-bore chest tubes. Operative cultures of the peel and the gelatinous exudate were negative in 8 patients. In the other 2 patients (patients 1 and 2), the exudate culture was similar to the preoperative pleural fluid culture.

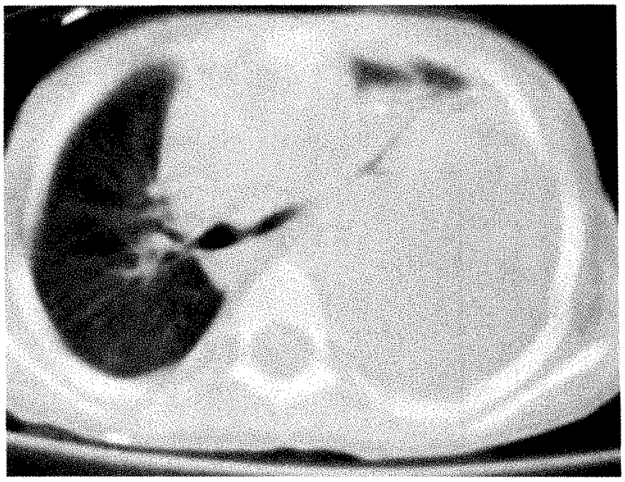

Fig 5. (Patient 3.) A computed tomographic scan of the chest reveals a large amount of fibrinous debris, which was almost completely collapsing the left lung and was not improved by pleural drainage.

Results

Recovery in all 10 patients was rapid. The fever subsided within 48 hours. The white blood cell counts returned to normal over four to seven days. All children were weaned from the ventilator and extubated by the second postoperative day; arterial blood gases were excellent. Signs of systemic toxicity resolved quickly. No patient had a persistent air leak, postoperative bleeding, or chylothorax. No child required a chest tube beyond the sixth postoperative day. Follow-up chest roentgenograms confirmed complete lung reexpansion. Antibiotic administration was usually continued for seven days after operation. The patients were discharged from the hospital an average of ten days after operation.

All patients have been followed in the thoracic surgery clinic. There is no evidence of scoliosis, recurrent pleural or parenchymal disease, restricted chest wall movement, or respiratory complaints (Fig 9).

Comments

In the past few decades, there has been a trend toward early decortication for posttraumatic empyema, with some authors [10, 11] recommending thoracotomy as early as seven to ten days after injury, if pleural drainage is failing. However, there has been some reluctance to proceed with early decortication for postpneumonic empyema because many authors [4, 5] believe that most patients recover eventually with pleural drainage. Bryant and colleagues [12] noted failure of intercostal tube drainage in 5 of 15 children with postpneumonic empyema within 48 to 92 hours after initiation of conventional treatment, yet delayed decortication an average of 28 days. Sensenig and co-workers [13] in 1963 sharply disagreed with prolonged conventional treatment: "Decortication is particularly appealing for use in the pediatric age group, in which prolonged tube drainage is avoided, scoliosis is prevented, and the patient rapidly returned to health." Since then, others [3, 7, 8] have pointed to the immense benefits of early decortication in select children

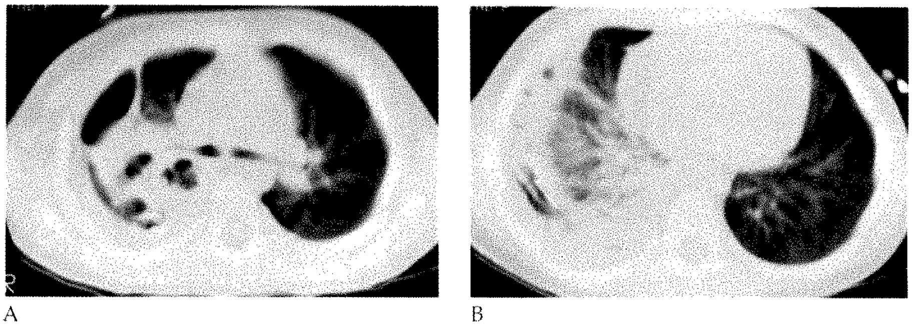

Fig 6. (Patient 6.) (A, B) Two computed tomographic scans of the chest show an extensive pleural peel entrapping the lung, with multiple loculations of fibrinous debris and a loculated pneumothorax. (A) The fibrotic process can be seen extending into the interlobar fissure.

who failed to respond to conventional therapy: no deaths and minimal morbidity from the decortication procedure. These differences reflect not only different philosophies (aggressive versus conservative) but more importantly, different patient populations.

Treatment considerations should be based on the stage of the disease, the particular bacteria isolated, the response to initial treatment, and the degree of lung trapping [7]. The exudative and early fibropurulent phases of an empyema can usually be treated by antibiotics and repeated thoracentesis or, more preferably, closed chest-tube drainage [9]. Often, repeated thoracentesis only partially and intermittently reexpands the lung. Sometimes an additional chest tube is necessary to completely evacuate the hemithorax and reexpand the lung. In the late fibropurulent and organizing phases, prolonged pleural drainage is often inadequate. None of the patients in this series was seen at our institution in the exudative or early fibropurulent phase. Considering the uniform operative findings of a major pleural peel and entrapped lung, no patient would have benefited from a longer trial of pleural drainage.

In the era before antibiotics, pneumococci and streptococci were the most frequent organisms isolated in postpneumonic empyemas. With the advent of penicillin, the incidence of pneumococcal and streptococcal empyema dramatically decreased and *Staphylococcus aureus* became the most commonly isolated bacteria [14, 15]. Empyemas caused by *S aureus* almost always respond well to conventional treatment and almost never require decortication. Series reporting excellent results with nonoperative management are heavily weighted with staphylococcal empyemas [2, 4]. The minithoracotomy and suction technique of Raffensperger and colleagues [6] was successful primarily in empyemas caused by *S aureus*.

The emergence of *H influenzae*, β-hemolytic streptococci, *S pneumoniae*, and anaerobes as common bacterial isolates has altered the pathophysiology of childhood empyemas [7, 8]. In sharp contrast to the unilocular staphylococcal empyema, these bacteria often cause multiple fluid loculations and a thick fibrous peel, which are not amenable to conventional therapy. In 6 of our patients β-hemolytic streptococci, *S pneumoniae*, and *H influenzae* were isolated. No bacteria could be isolated in 4 patients. The operative findings of multiple loculations and fibrous pleural peel were impressive in all 10 patients.

The widespread use of antibiotics probably aborts a number of early pleural infections. With appropriate antimicrobial treatment, the incidence of empyema after pneumonia has decreased. Still, the success of conventional therapy depends to a great extent on the stage of empyema development at presentation. Therefore, failure to recognize an early, developing empyema with delay in establishing pleural drainage and ineffective or inappropriate antibiotic therapy allows progression of the empyema process. The child's symptoms are often suppressed. The unrecognized fibropurulent material organizes, and the lung becomes entrapped. All patients in this series had initially been treated with multiple antibiotics without early recognition of the pleural process and a thoracentesis to obtain a pleural fluid culture. Adequate and aggressive pleural drainage had not been established in the exudative or early fibropurulent phase in any patient before transfer.

The clinical response of patients to appropriate antibiotics and adequate pleural drainage is very important. If the child is steadily improving, treatment should be continued. Conversely, if the patient is having respiratory difficulty, daily fevers, and a persistent leukocytosis, he or she should be considered for early lung decortication.

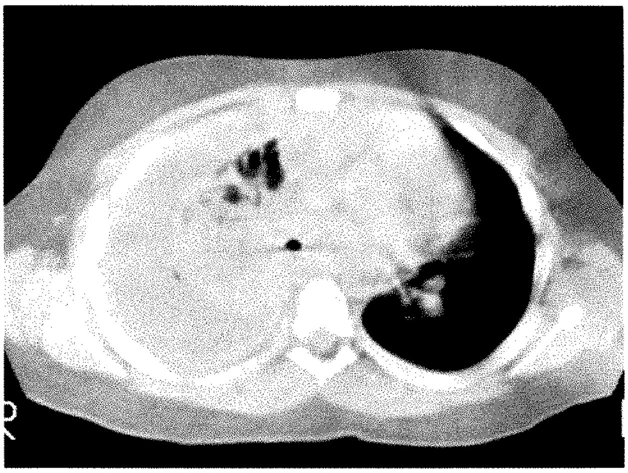

Fig 7. (Patient 4.) Computed tomographic scan of the chest shows large accumulation of fibrin coagulum compressing the right lung. Cystic changes in the right upper lobe are visible.

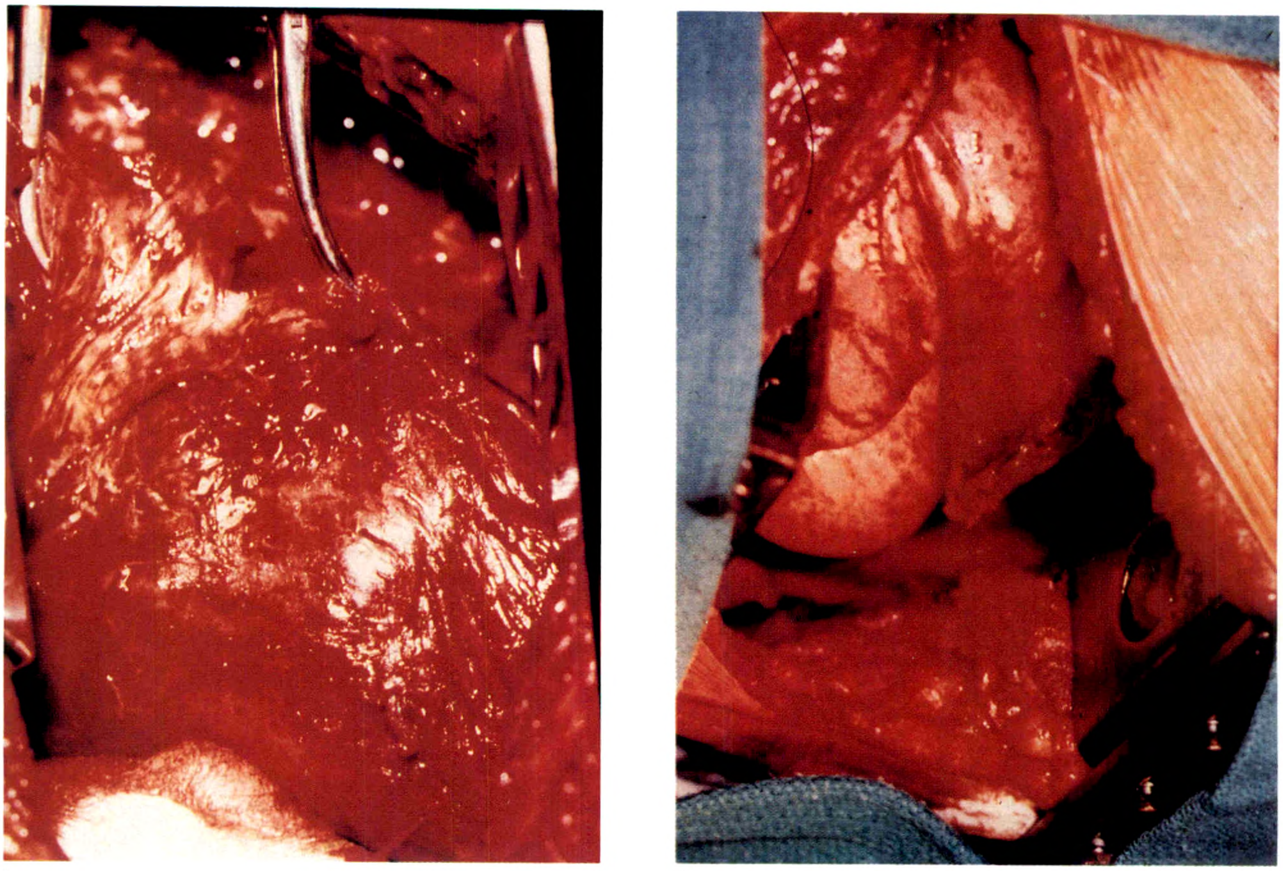

Fig 8. (A) Rankin's clamps were placed on an extensive pleural peel encasing the right lung after evacuation of the fibrinous exudate. (B) The peel has been stripped from the visceral pleura and both fissures, allowing complete reexpansion of the right lung.

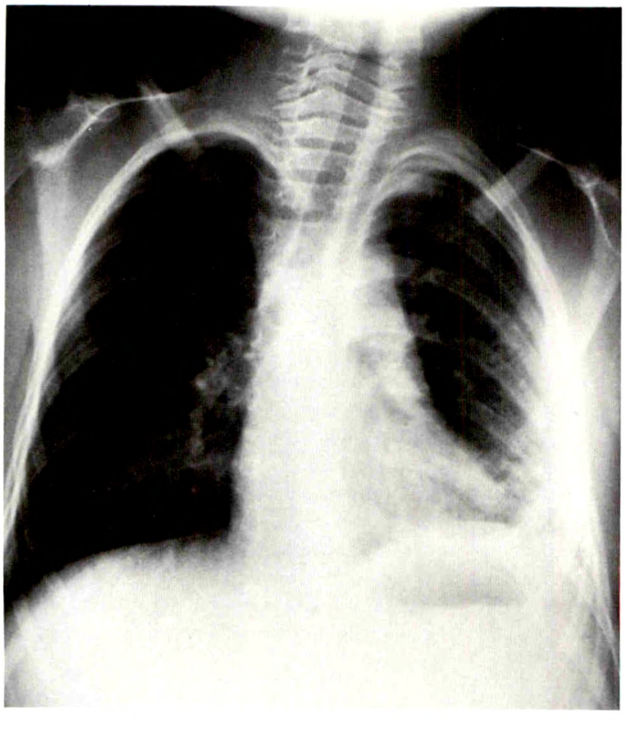

Fig 9. (Patient 7.) A follow-up chest roentgenogram made 2 years after decortication demonstrates complete reexpansion of the left lung with only minimal pleural reaction.

Even when continued pleural drainage is eventually successful, there may be a prolonged period of fever and toxicity. Lung trapping causes atelectasis, which often is associated with daily fevers. The greater the toxicity, the more urgent the need for decortication. Six patients in this series had negative pleural cultures. Even so, their physiological response to the empyema was equally severe, confirming the part played by the fixed thoracic wall, compressed lung, and intrapleural debris in the toxicity and morbidity of an empyema. When the empyema reaches the organizing stage, there is little excuse for waiting weeks to see if some resolution will occur. Prolonged pleural drainage in these patients may even be detrimental. Particularly in young patients, persistent lung compression can adversely affect lung growth and pulmonary function, as lung growth and alveolar development continue until 3 years of age.

It is important to realize that the chest roentgenogram underestimates the extent of pleural disease and the volume of lung entrapped by the pleural peel. Chest computed tomography has been shown to be valuable in the evaluation of postpneumonic empyema by delineating pleural disease and lung trapping from parenchymal disease [16]. The chest CT scans were especially helpful in our patients who had complete opacification of a hemithorax. In 2 patient, the CT scans demonstrated a coexisting pneumonia with cystic changes. When the CT scan showed multiple loculations of fibrin coagulum compressing the lung (4 patients) or an extensive pleural peel entrapping the lung (4 patients), a decision to proceed with early decortication was strongly considered. The operative findings correlated directly with the chest CT scan in every instance.

A relatively small percentage of children treated by aggressive conventional therapy can have continued symptoms of infection and pulmonary compromise. In our patients, a critical delay in diagnosis and a delay in initiating appropriate antibiotic therapy and adequate pleural drainage could have contributed to the failure of conventional therapy. Preoperative preparation should be held to a minimum once conventional therapy failure is determined. Several of our patients were operated on within 48 to 72 hours after transfer. Early decortication in this series of refractory, symptomatic empyemas resulted in immediate and complete reexpansion of the entire lung, with minimal morbidity and no deaths. Clinical improvement was marked, the child's hospital stay was shortened, and the risks of long-term antibiotics and pleural drainage were minimized. Decortication carried out at a later time is often associated with much more bleeding from the pleura and more air leaks from the lung surface, leading to a more complicated postoperative recovery.

In summary, early lung decortication should be considered in select children with postpneumonic empyema whose disease is caused by bacteria other than *S aureus* and whose clinical course does not improve. A chest CT scan showing multiple loculations of fibrinous debris or an extensive pleural peel entrapping the lung can be very helpful in the decision-making process in these sick children. Complete reexpansion of the lung by early decortication can substantially alter the natural history of the disease in these symptomatic children. The final role of decortication in childhood empyemas has yet to be determined, but the patients in this study had complete recovery and essentially normal chest roentgenograms over follow-up, yielding excellent long-term results.

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DISCUSSION

DR JOHN J. WHITE (Loma Linda, CA): At Loma Linda University, my colleagues and I have been interested in optimizing our care of children with empyema, and we agree with the observations presented. We have reviewed the cases of all such patients seen in the past 9 years, and not just those who had decortication. We have treated 27 children with an average age of 6½ years. Twenty-five children have been followed up; 2 have moved out of the state.

We resorted to decortication in only 7 patients. We were able to treat 5 with appropriate antibiotics as determined from the results of thoracentesis only; 15 required tube thoracostomy; 5 had persistent disease and went on to decortication; and 2 required concomitant lung resection because of lung disease and abscess formation.

Twenty-four children are alive; there was one late death not associated with the empyema. There have been no recurrences, and full activity is enjoyed by all of our patients.

From our experience, I stress two points: early treatment of empyema and appropriate antibiotic treatment with proper drainage. Those children who do not respond and have a downhill course merit early decortication. I caution that we must not be ruled by the powerful images obtained using the CT scan. I have been approached on a number of occasions by pediatric colleagues who want me to operate on a very healthy, afebrile, and active child who happens to have loculations in the pleural space. I have operated on none of these children, and all have turned out well. The overriding principle in treating empyema is to treat the child, not the CT scan.

DR BRADLEY M. ROGERS (Charlottesville, VA): This is a beautifully prepared and very well illustrated presentation. I think we all would agree that the need for decortication in these patients merely reflects a failure of early therapy.

My co-workers and I recently reviewed our experience over the past 17 years. We have never had to use decortication in a child with empyema. However, I think we tend to be much more aggressive in the early evacuation of the chest.

I stress the early use of chest tubes, and the use of intrapleural streptokinase. In some of the patients with chest CT scans such as you have shown, the aggressive use of thoracoscopy to break down the locules and wash out the pleural space is very, very effective. This can be performed under local anesthesia and allows debridement of the entire pleural space with a very simple procedure. I think we should be able to eliminate the need for this operation completely if we attack the process in these children early.

DR J. KENT TRINKLE (San Antonio, TX): Pediatric empyema is treated rather poorly, probably because it is treated initially by pediatricians rather than surgeons. At a referral institution like yours, you see the treatment failures. Conventional wisdom says empyema can be treated with antibiotics and thoracentesis; rarely is it necessary to use a tube and never should a child's chest be touched with a knife. Conversely, in adults, a tube is always used and frequently decortication. I think this is not the difference between adults and children, but rather the fact that children tend to get effusions, which stay liquid for a long time and can be evacuated, after viral or gram-positive pneumonia.

I know you had the bacteriological results, and I know you had experienced the failures of so-called conservative treatment. Do you have any correlation between the bacteriological findings and the failures? Finally, one has to remember that if the empyema reaches the fibropurulent or the peel stage, a fibrothorax develops. As the child grows, that side of the chest is not going to keep up, and a kyphosis is the result later in life. That is the real tragedy of neglected empyema.

DR J. ALEX HALLER (Baltimore, MD): The comment about failure of pediatricians to begin more aggressive early treatment has been made by others, but I think this may be a failure on our part as well, because much of the literature is filled with the early management of staphylococcal empyemas. As many of us remember, such empyemas rarely require decortication. We taught our pediatricians well in that era, but now most of them believe that you do not have to have a surgeon involved in the initial management of all childhood empyemas. I think we need to change that perception. Any septic effusion, that is, one that has bacteria at the time of the initial tap, should have thoracostomy tube draining.

What do you think about that? Do your pediatricians in West Virginia accept this concept?

DR MILTON V. DAVIS (Kaufman, TX): Respectfully, I disagree with the concept of this paper by Dr Gustafson and the others from West Virginia. The title is questionable. I do not want to make a big point about a word, but this probably could better be called a gelatinous pleuritis than an empyema. The fact that a positive culture is or is not grown is certainly helpful and not ever to be discouraged, but sometimes positive cultures result from something that is not an active infection. The results of your treatment are good, and I cannot fault them. However, these children, for the most part, really will get well without an open operation.

There are two points I really want to challenge. First, your point that because the child is in trouble as a result of something that is happening on one side of the chest and requires a ventilator, thus dictating that we should clean out the pleura on that side, is simply physiologically not valid. You can take out a whole lung, and the child will do all right if the other lung is working properly. Second, I challenge the interpretation of the CT scans and the roentgenograms. It simply does not coincide with the lung experience of many of us.

DR GUSTAFSON: Certainly we believe that one should operate predominantly on the basis of symptoms and not the chest roentgenographic or CT scan findings. The patients in this series were very symptomatic. The 2 patients who were on a ventilator preoperatively were operated on more for sepsis than they were for the impact of the empyema on their pulmonary status. They were adequately managed on the ventilator, but they could not be weaned from it. These 2 patients had positive blood cultures, and 1 also had a positive pleural fluid culture.

We have not used thoracoscopy or infusion of streptokinase, because we believe that the risk of decortication is very low.

We agree there is a problem in educating pediatricians about the current management of postpneumonic empyema. Pediatricians often refer to literature about staphylococcal empyema, which suggests that surgical intervention is rarely necessary. In no patient in this series was *S aureus* isolated. None of these patients were initially managed by pediatricians at our institution; they were all referral patients. I think the bias of the referring pediatricians was that their patients would get well without aggressive therapy. After a number of days and sometimes weeks of antibiotic therapy, the patients were then transferred, when they were quite sick. After a short trial of pleural drainage and appropriate antibiotics failed to improve their clinical status, we decided early decortication was an appropriate option to resolve the empyema. I think there is a need to educate pediatricians about modern-day empyema.

Long-Term Results of Direct Aortoplasty for Repair of Aortic Coarctation in Adults

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The long-term results of 14 adult patients who underwent a direct aortoplasty for aortic coarctation were reviewed. The mean age of the patients at operation was 31.5 years. Three of the patients died; 1 died perioperatively, and 2 died later of cardiovascular disease. The survivors were followed for 15.8 years. None of the patients had complications at the site of coarctation repair. This contrasts with our earlier findings on patch graft aortoplasties, which showed a high incidence of postoperative aneurysm at the repair site. The etiology of these aneurysms is discussed with reference to the information provided by the present study.

(Ann Thorac Surg 1990;49:948–50)

Direct aortoplasty or isthmoplasty consists of widening the stenosed segment of the coarctation without resection of the aorta or resorting to use of synthetic material [1]. If the stenosis is short and the aorta is not atheromatous or heavily calcified, this type of repair can be considered for select patients. The technical advantage of the method is that less surgical dissection is needed to widen the coarctated aorta than in resection of aorta. Indirect isthmoplasty with synthetic patching has been associated with late aneurysm development at the operative site [2–5]. Direct isthmoplasty as a standard technique in coarctation repair is rarely performed, and little information is available about the long-term results of patients who have undergone this procedure [2, 4, 6]. We present an analysis of our long-term results with direct aortoplasty and discuss the possible mechanisms behind the late complications of the indirect method.

For editorial comment, see page 863.

Material and Methods

Between 1967 and 1979, direct aortoplasty was performed for aortic coarctation repair in 14 adult patients at Helsinki University Central Hospital. The patients ranged in age from 16 to 51 years (mean age, 31.5 years) at operation. Nine of the patients were men and 5 were women. The ages of the men ranged from 17 to 51 years, and those of the women ranged from 16 to 48 years. The aorta was exposed through a left posterolateral thoracotomy under general anesthesia. After proximal and distal cross-clamping of the coarctated segment, the intercostal arteries were divided if necessary. In 13 patients, aortoplasty was performed by incising the stenotic segment longitudinally with transverse closure. In 1 patient, an intercostal artery was used as additional material. In another patient, the plasty was reinforced with a Dacron prosthetic support placed around the repaired site. The aorta was closed with continuous nonabsorbable synthetic suture material. Aortography was performed in the patients in 1980. Thereafter, the cardiovascular state was noted and chest roentgenograms were taken regularly.

Results

One of the patients did not survive the operation. The death resulted from intractable hemostatic problems. Thus, the surgical mortality in this series was 7.1%. Two patients died of cardiovascular complications during the follow-up at 50 and 51 years of age; 1 died 5 years and the other died 17 years after the aortoplasty. The cause of death was ischemic myocardial attack in both patients. The survivors (11 patients) have now been followed for an average of 15.8 years (range, 9 to 21 years). One patient has ischemic heart disease, and 3 have high blood pressure controlled by medication. Two of the patients have undergone aortic valve replacement, one 10 years and the other 16 years after the coarctation repair. In addition, aortic valvular disease has been diagnosed in 3 other patients, so far treated nonoperatively. The mean age of the survivors was 46.9 years (range, 31 to 71 years) at the latest follow-up examination in 1988.

Angiographic examination showed no specific changes at the operative sites. Later, any enlargement of the mediastinum of more than 1 cm, as observed in chest roentgenograms, was considered clinically significant, necessitating further examinations. There were 3 such cases. Aortography showed a dilated ascending aorta in 1 patient 9 years after the coarctation repair. Computed tomographic scans performed on 2 patients showed no pathological features in the aorta. In the other patients, the cardiovascular state and chest roentgenograms showed no clinically significant changes. There was no recurrent coarctation as judged from an examination of the hemodynamic state, which included blood pressure measurements on the lower extremities. It was notewor-

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Table 1. Late Follow-up and Associated Lesions of the Survivors

Sex	Age at Operation (yr)	Follow-up (yr)	Associated Lesions
F	26	9	Dilated ascending aorta
M	34	10	Aortic valve disease and hypertension
M	37	21	Ischemic heart disease
M	16	18	Aortic valve repair
M	18	13	None
F	48	18	Aortic valve repair
M	36	18	Aortic valve disease and hypertension
M	21	18	None
F	17	20	None
F	51	20	Aortic valve disease and hypertension
M	26	9	None

thy that in none of the patients did aneurysms develop at the site of coarctation repair. The survivors' associated lesions are shown in Table 1.

Comment

Resection of the stenosed segment with end-to-end anastomosis is the technique most widely used in repair of adult coarctation. With the exceptions of vascular tubular graft interposition and aortoplasty using the subclavian artery, other methods are used only rarely [2, 6–8]. Direct aortoplasty offers an alternative method of treating this congenital anomaly in selected cases, however, without resorting to use of synthetic material. Indeed, the presence of abnormal or ductal tissue typically left in situ in operations of this type has not induced subsequent postoperative aneurysms such as have been attributed to the Dacron patch onlay method [4, 9, 10]. We have observed no tendency to enlargement or aneurysm development at the repair sites in patients subjected to direct isthmoplasty. This contrasts with the high incidence of late aneurysms experienced with the indirect patch onlay method [3–5].

The pathogenesis of aneurysm formation is not yet clearly understood. Several mechanisms have been proposed as responsible factors, including congenital weakness of the aortic wall, extensive resectioning of the aortic intima, the presence of ductal tissue at the repair site and abnormal tension caused by the rigidity of the prosthetic material against the aortic wall [3, 4, 9–11]. Despite resectioning of the aortic intima, the ductal tissue in the aortic wall appears to resist new hemodynamic forces resulting from the repair. This observation offers support for the hypothesis that aneurysms originate from lack of compliance of the synthetic rigid material implanted into the aorta. The compliance of Dacron is about 1% of that of the normal aorta [12]; however, Dacron and other synthetic materials have been widely used to dilate arterial stenosis elsewhere in arteries of smaller size without such a high occurrence of late aneurysms. The aneurysms that occurred after synthetic patch onlay aortoplasty were true aneurysms, not false aneurysms such as originate from suture lines in other arteries (eg, in femoral arteries).

Associated lesions were frequent. The incidence of aortic valve disease was 36%. This high frequency is in accordance with previous reports in which congenital aortic valve disease has often been found to coexist with coarctation [13, 14]. Some of these patients also have hypertension that might have accelerated the rate of aortic valvular degeneration. Irreversible hypertension has often developed when the coarctectomy has been delayed until adulthood [7, 8, 13, 14]. Long-standing high blood pressure is also an important etiological factor in the increased incidence of ischemic heart disease [6, 15]. In this series, late postoperative deaths resulted from acute myocardial infarction.

The optimal time for repair of aortic coarctation is before the patient reaches school age. If the diagnosis is delayed until adulthood, however, resectioning the coarctation with end-to-end anastomosis is a valid method. In select patients, direct isthmoplasty is an alternative worth considering because of its technical simplicity. The long-term results are satisfactory, and we have observed no subsequent negative consequences related to the technique itself.

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Notice From the American Board of Thoracic Surgery

The American Board of Thoracic Surgery began its recertification process in 1984. Diplomates interested in participating in this examination should maintain a documented list of the operations they performed during the year prior to application for recertification. They should also keep a record of their attendance at thoracic surgical meetings, and other continuing medical education activities, for the 2 years prior to application. A minimum of 100 hours of approved CME activity is required.

In place of a cognitive examination, candidates for recertification will be required to complete both the general thoracic and cardiac portions of the SESATS III syllabus (Self-Education/Self-Assessment in Thoracic Surgery). It is not necessary for candidates to purchase SESATS III booklets prior to applying for recertification. SESATS III booklets will be forwarded to candidates after their applications have been accepted.

Diplomates whose 10-year certificates will expire in 1992 may begin the recertification process in 1990. This new certificate will be dated 10 years from the time of expiration of the original certificate. Recertification is also open to any diplomate with an unlimited certificate and will in no way affect the validity of the original certificate.

The deadline for submission of applications is July 1, 1990. A recertification brochure outlining the rules and requirements for recertification in thoracic surgery is available upon request from the American Board of Thoracic Surgery, One Rotary Center, Suite 803, Evanston, IL 60201.

Tracheal Reconstruction by Esophageal Interposition: An Experimental Study

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The purpose of this study was to assess the possibility of reconstructing a circumferential tracheal defect with autogenous esophagus. In 6 mongrel dogs, a circumferential defect involving seven rings of the cervical trachea was reconstructed by interposing pedicled esophagus. A silicone T tube was used as a stent. The vertical limb of the T tube (usually referred to as the horizontal limb when used in humans) was cut shorter after the cervical wound had healed well, and it eventually was buried subcutaneously. Two dogs died 36 days after operation, and 1 died 28 weeks after operation. In no dog was the cause of death related to the operation or to a respiratory tract complication. Two dogs were put to death 4 weeks and 32 weeks after operation. They were well until then, and all the anastomoses between the trachea and the esophagus had healed fully without formation of granulation tissue. One dog is alive and well 14 months after operation. The vertical limb of the T tube retracted into the subcutaneous space, and there is no open cervical wound. Esophageal interposition might be a feasible technique for tracheal replacement in select groups of patients.

(Ann Thorac Surg 1990;49:951–4)

Most tracheal defects can be repaired primarily by end-to-end anastomosis. Some, however, cannot [1] because too much of the trachea must be resected or because adequate tracheal mobilization is not possible. The purpose of this study was to assess the possibility of reconstructing the trachea with autogenous esophagus.

Material and Methods

Six adult mongrel dogs weighing 10 to 15 kg were used in the study. Anesthesia was induced with intramuscular administration of ketamine hydrochloride and maintained by intravenous administration of sodium thiopental. Perioperatively 1 g of cefamandole naftate was given intravenously, and the wound was lavaged with 90 mg of tobramycin sulfate just before closure. Ventilation was maintained by spontaneous breathing throughout the operation. No antibiotics were given postoperatively.

For editorial comment, see page 864.

After the animal was prepared with povidone-iodine solution and draped, a midline cervical incision was performed. Cervical strap muscles were divided to expose the trachea and esophagus. A segment of trachea including seven tracheal rings (rings 8 through 14) was dissected and resected circumferentially. The connective tissue posterior to the esophagus adjacent to the resected trachea was preserved as the pedicle. The esophagus proximal and distal to this segment was mobilized by sharp and blunt dissection for esophageal reconstruction by end-to-end anastomosis.

The esophagus was transected at the levels of tracheal transection (Fig 1A). The lumen of the esophagus was scrubbed with povidone-iodine solution. End-to-end anastomosis of the esophagus was done first by interrupted vertical mattress sutures of 3-0 braided polyglycolic acid (Fig 1B). Then the posterior half of the proximal and distal tracheoesophageal anastomoses was done by interrupted whole-layer tracheal and musculoadventitial esophageal sutures of 3-0 braided polyglycolic acid.

A silicone T tube (outer diameter, 10 mm; inner diameter, 8 mm; proximal horizontal limb, 4 cm; distal horizontal limb, 4 cm; vertical limb, 4 cm) was inserted into the interposed esophageal lumen. The vertical limb (usually referred to as the horizontal limb when used in humans) of the T tube was brought out through a stab wound created in the anterior wall of the interposed esophagus. Finally, after the lumen of the interposed esophagus was supported with the T tube, the anterior half of the proximal and distal tracheoesophageal anastomoses was completed (Figs 1C, 2).

The strap muscles were reapproximated, and the skin was closed with the vertical limb of the T tube protruding through the cervical wound. The vertical limb of the tube was corked after tracheobronchial suction.

The dogs were returned to their cages on the day of the operation and from the next day on, were given regular dog food and water. About 2 months after operation, when the wound had healed well, approximately 1 cm of the vertical limb of the T tube was pulled out and cut at the level of the surface of the skin. The vertical limb

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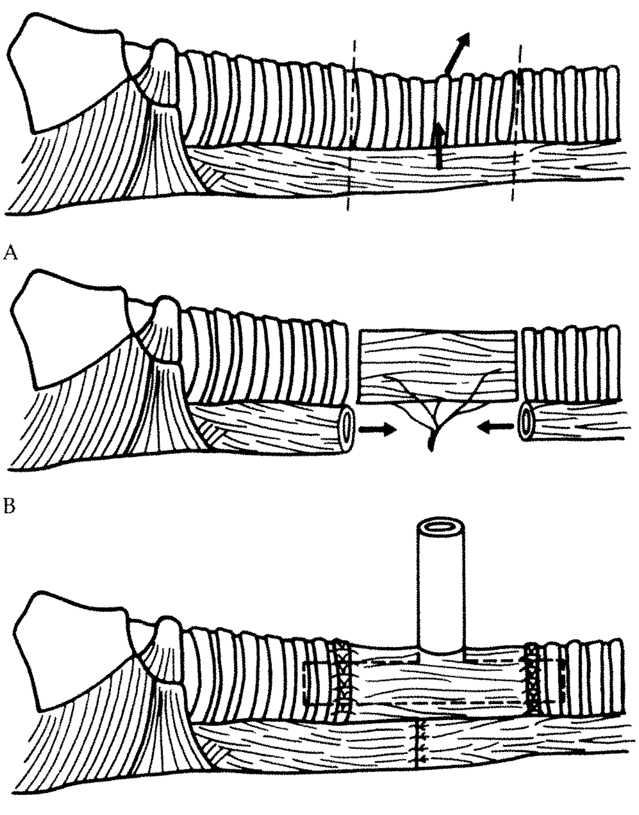

Fig 1. (A) Seven rings of the trachea (rings 8 through 14) were circumferentially resected, and the esophagus was transected at the levels just distal to the seventh and 14th rings for tracheal replacement. (B) The esophagus was reconstructed by end-to-end anastomosis after mobilization. A pedicled segment of esophagus was brought forward for tracheal reconstruction. (C) The trachea was reconstructed with interposed esophagus and a silicone T-tube stent.

retracted into the subcutaneous space. Two dogs were put to death for macroscopic and microscopic observations. Specimens for microscopic observation were stained with hematoxylin and eosin. One dog has been kept alive for long-term observation. All the animals received humane care throughout the study.

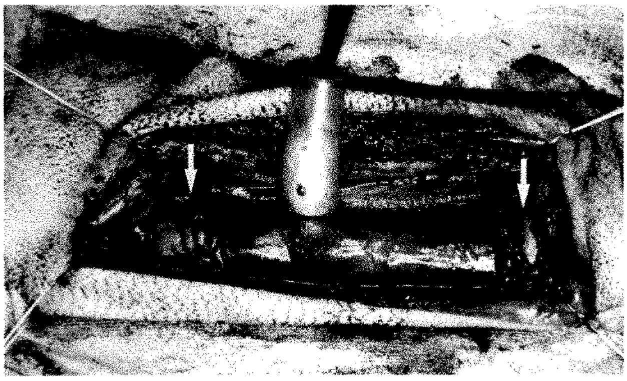

Fig 2. Operative view. Tracheoesophageal end-to-end anastomoses were done by interrupted sutures of 3-0 braided polyglycolic acid. A silicone T tube was inserted in the interposed esophagus, and the vertical limb (horizontal limb when placed in humans) was brought out through a stab wound created in the anterior wall. (arrows = *anastomoses.)*

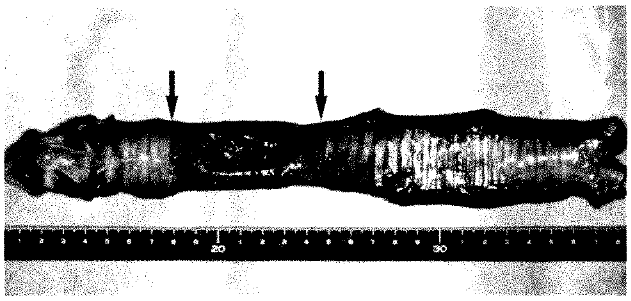

Fig 3. External view of the trachea and interposed esophagus of the dog killed 32 weeks after operation. (arrows = *anastomoses.)*

Results

All dogs survived the operation. Cough was present in every animal for about 1 month postoperatively, but it subsided as the time after operation lengthened.

Three dogs died before they were killed. One died of severe enteritis with melena 36 days after operation. The other 2 were in good condition until they died suddenly, one 36 days after operation and the other 28 weeks after operation. The cause of death in these 2 dogs could not be specified. In every dog, postmortem examination revealed good healing of all three anastomoses without anastomotic stenosis or signs of infection.

Two dogs were put to death 4 weeks and 32 weeks after operation for macroscopic and microscopic observations. Both proximal and distal tracheoesophageal anastomoses had healed well (Figs 3, 4), and continuity of the epithelium had been established (Fig 5). There was no formation of granulation tissue at the anastomoses. In the distal

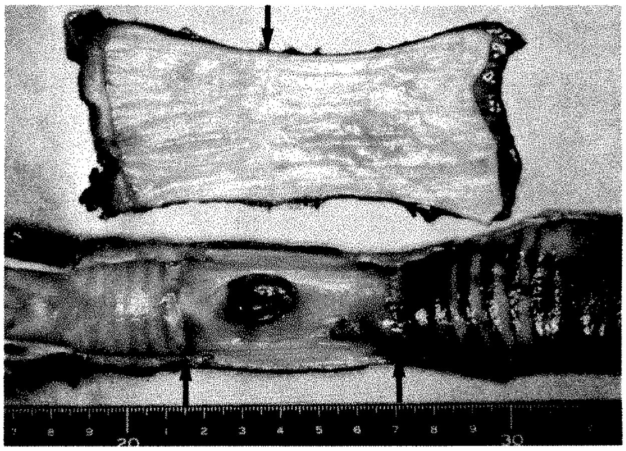

Fig 4. Internal view of the interposed esophagus (bottom) and reconstructed esophagus (top) of the dog killed 32 weeks after operation. The trachea and the interposed esophagus were opened in the posterior wall. All anastomoses had healed well, and the anastomotic lines had epithelialized. The T tube was kept in place by the vertical limb, which stretched out through the hole in the center of the interposed esophagus. The proximal portions are to the left and the distal portions, to the right. (arrows = *anastomoses.)*

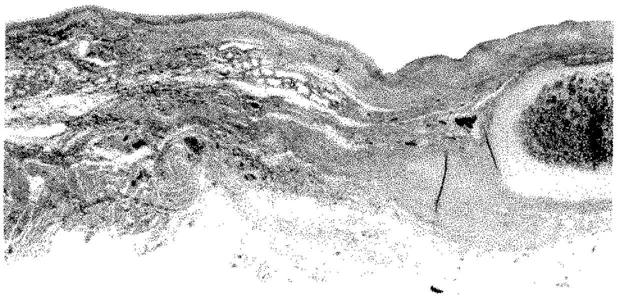

Fig 5. Microscopic view of tracheoesophageal anastomosis. Continuity of the epithelium had been restored. Squamous cell metaplasia of the tracheal epithelium was present. The interposed esophagus is to the left and the distal trachea, to the right. (Hematoxylin and eosin; ×50 before 52% reduction.)

trachea of the dog killed 4 weeks after operation, erosion was present at the site where the T tube was in contact with the tracheal wall. The esophagoesophageal anastomosis had healed well without stenosis in both dogs (see Fig 4).

One dog is alive and in good condition 14 months after operation. The tracheostomy (or interposed esophagostomy) closed after the vertical limb of the T tube was shortened and buried subcutaneously.

Comment

Many attempts at tracheal replacement have been made experimentally and clinically. Methods for tracheal replacement can be classified into three categories: replacement by artificial materials [2–4], replacement by tracheal homograft [5–8], and replacement by autogenous materials [9–13]. When an artificial trachea is used, there always seems to be the danger of anastomotic dehiscence and migration of the prosthesis because there is no wound healing process between the host tissue and the prosthesis. Replacement by homograft implies the problem of blood supply to the graft and immunosuppression. Viability of the tracheal cartilage of the graft appears to be critical for the success of the homograft [7, 8]. The use of autogenous material could leave an anatomical or functional defect in the organ utilized. The method presented here belongs partially to the first category and partially to the last category, as continuity of the airway is maintained by autogenous material and patency of the airway is maintained by artificial material.

When the trachea is reconstructed with autogenous tissue, the esophagus seems to be one of the most adequate organs to use for three main reasons. First, no complicated surgical technique is necessary for its interposition because it lies right next to the trachea in its full extent. Second, its lumen is lined with epithelium. Third, there are established techniques for esophageal reconstruction in humans. There appear to be few problems in using a silicone T tube as a stent, as it is already clinically used as a stent to keep the tracheal lumen patent for long periods [14]. The vertical limb of the T tube can be safely buried subcutaneously, as some clinical experiences [15, 16] indicate.

There are some reports of emergency cases of congenital tracheal aplasia or stenosis in which the esophagus with a stent was used as an airway [17, 18]. The results were not satisfactory. We do not believe that these reports preclude the use of the esophagus as an airway, though further study is needed before clinical use.

In the present study, we only partially replaced the cervical trachea with esophagus and reconstructed the esophagus by end-to-end anastomosis. This technique could be used only in reconstructing rather short tracheal defects that could usually be reconstructed by end-to-end anastomosis after tracheal mobilization. Clinically the technique might be used when adequate tracheal mobilization is not possible, as in a reoperation or in the case of a trachea with little elasticity.

In the replacement of a longer tracheal defect that cannot be repaired by direct anastomosis even after mobilization, reconstructing the esophagus by end-to-end anastomosis may be impossible. In this case, an esophageal bypass procedure, which could not be performed in the dog because of its larger proportion of thorax to abdomen compared with a human, must be added to reconstruct the esophagus.

We conclude that a tracheal defect might be reconstructed by interposing esophagus in select patients who might otherwise be treated by terminal mediastinal tracheostomy or insufficient resection. The indications for our procedure may be limited, however.

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REVIEW OF RECENT BOOKS

Diagnosis of Diseases of the Chest, 3rd Edition, Volume II
By Robert G. Fraser, J. A. Peter Paré, P. D. Paré, Richard S. Fraser, and George P. Genereux
Philadelphia: WB Saunders, 1989
1,772 pp, illustrated

Reviewed by Darroch W. O. Moores, MD

It was indeed a pleasure to review this textbook. It provides a tremendous education for all students of pulmonary disease regardless of discipline or level. This textbook continues to be the most widely read and respected textbook of its kind in the English-language literature. Volume II includes developmental anomalies, infectious disease, immunological disease, and neoplastic disease of the lungs.

The section on neoplastic disease of the lung (chapter 8, pages 1,327 to 1,623) is a very extensive and complete review of both benign and malignant neoplasms arising in the lungs. Primary bronchogenic carcinoma as well as neoplasms of the tracheobronchial glands, leukemia, lymphoma, sarcoma, and other miscellaneous tumors are included. The chapter focuses on the diagnosis of these tumors rather than therapy. The radiological illustrations throughout the book are exquisite and are unequalled. The pathology plates, both gross and histological, are excellent. The work and detail that has gone into the preparation of this book is to be commended.

This textbook is at the interface between pulmonary medicine, thoracic surgery, and pulmonary radiology, and should sit in the personal library of all thoracic surgeons.

Albany, New York

Experimental Reconstruction of the Canine Trachea With a Free Revascularized Small Bowel Graft

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Extensive tracheal stenotic lesions caused by tracheomalacia or neoplasms represent a surgical challenge. Segmental tracheal substitution is sometimes required to obtain radical cure. We present an experimental study of 27 dogs undergoing replacement of the cervical trachea using a vascularized small bowel segment as a tubular graft. A silicone stent was placed in the lumen of the intestinal fragment and was removed the second week after operation. Endoscopic and histological examinations were performed between the first week and second month after operation, and rigidity of the graft was assessed in all cases. No evidence of anastomotic stricture or mucous formation was found. Microscopic examination showed the substitution of bowel mucosa by squamous epithelium as well as the development of connective tissue favoring the fixation of the skeletal muscular structures of the neck to the serous layer of the graft, thus avoiding collapse of the new airway.

(Ann Thorac Surg 1990;49:955–8)

Reconstruction of extensive tracheal lesions caused by stenosis, tracheomalacia, or neoplasms represents a surgical challenge. The advantages afforded by the use of autoplasties in tracheal reconstruction [1–9] as opposed to prostheses [10–14] are well documented. In some cases, however, the extent of the tracheal defect makes it impossible to use autogenous grafts.

Based on previous experience in pharyngoesophageal reconstruction [15, 16], it has been our aim [17] to find an autologous graft with its own vascular pedicle that (1) would be tubular, (2) could be surgically revascularized, (3) would be adaptable to the required length, and (4) would not collapse but rather would act as a rigid, elastic tube. In our experimental study, a segment of small bowel whose pedicle had been revascularized by anastomosis with cervical vessels was used as a tubular graft.

For editorial comment, see page 864.

Material and Methods

Twenty-seven mongrel dogs were included in the study. Operations were performed with the dogs under general anesthesia and endotracheal intubation (intravenous pancuronium bromide; fluothane 5%). The skin was prepared with a solution of povidone-iodine.

Surgical Procedure

A longitudinal midline abdominal incision, 10 cm in length, was made. Once a jejunal loop was selected, we performed as long a dissection of the vascular pedicle as possible. The intestinal loop was transected, preserving its vascularization, at the same time as end-to-end intestinal anastomosis was performed. The loop was gently handled and was kept moist by immersion in saline solution at 38°C. The average length of the resected intestinal segment was 12 cm (Fig 1).

A right anterior vertical cervical incision was made in front of the sternocleidomastoid muscle, and the external jugular vein (7 cm) and the common carotid artery (7 cm) were dissected and exposed. The intestinal vascular pedicle was sectioned, and sodium heparin was infused intraarterially. Then a microsurgical end-to-end anastomosis of the mesenteric artery to the common carotid artery was performed, with the latter temporarily occluded by an articulated clamp. A series of 7-0 Ti-cron U sutures was placed. The artery forceps was then released, and the correct venous reflux through the graft, together with its peristalsis, was verified. The mesenteric vein was anastomosed to the external jugular vein. The sternocleidomastoid muscle was partially sectioned, and the vascular pedicle was positioned to compensate for the difference in width between the jugular vein and the carotid artery.

The intestinal graft was preserved, and its correct vascularization and venous drainage were assessed. Subsequently, the tracheal segment was excised, 10 rings were removed, and the distal end was intubated with a curved tube (Mountandon cannula). The graft was prepared by washing the intestinal lumen and scraping the mucosa with a dry gauze. A silicone stent (endoprosthesis) was placed in the interior of the intestinal lumen (12 cm) projecting 2 cm at either end of the graft. The proximal end of the endoprosthesis was introduced into the proximal tracheal end and fixed in place by two absorbable sutures. The proximal tracheointestinal anastomosis using a series of 3-0 Vicryl U sutures was per-

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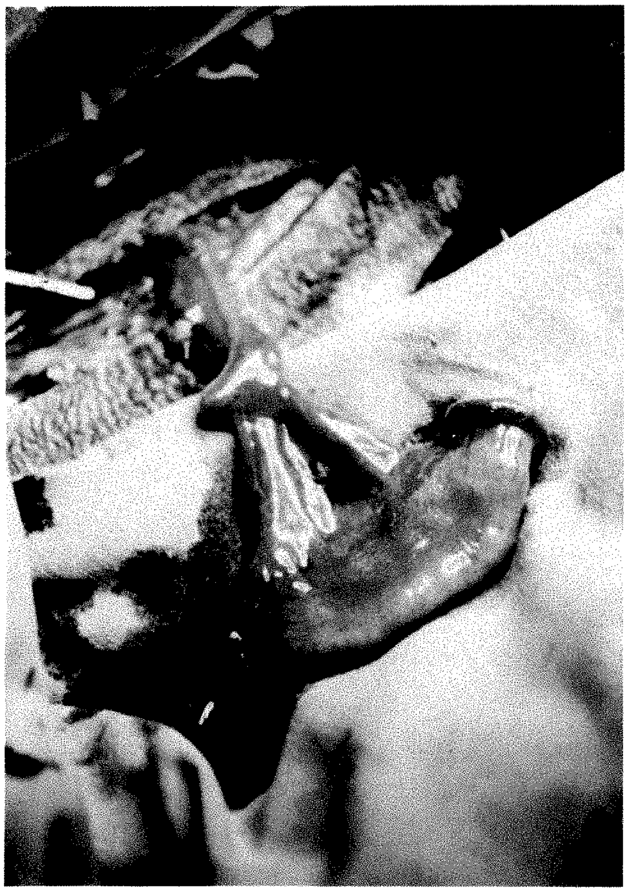

Fig 1. *Gross appearance of the resected intestinal loop with its vascular pedicle.*

formed. The Mountandon cannula was removed, and the endoprosthesis was introduced into the distal tracheal end; ventilation was instituted through the graft. Finally, the distal tracheointestinal suture was performed (Fig 2). The graft was covered with muscular structures of the neck, thus creating a homogeneous sheath without suturing the muscle directly to the intestinal serosa, and the wound was closed.

Postoperative Period

All dogs were extubated immediately after operation and 24 hours later were put on a liquid diet. Endoscopic examination was used for postoperative control during the second week after operation in the second series. In that series, the endoprosthesis was removed between days 10 and 14, and the success of the graft was assessed by studying the anastomoses and recording the presence or absence of malacia in the new airway, the reduction in the diameter of the neotrachea, and evidence of stenosis, granulomas, or mucous secretions. During the postoperative period, the following variables were monitored: ventilation, hypersecretion, diet, intestinal transit, urinary output, and behavior of the animal.

Histopathological examination particularly emphasized the tracheal regions proximal and distal to the graft, upper and lower tracheointestinal anastomoses, mesentery, transplanted intestinal wall, and the tissue surrounding it.

Results

All dogs survived operation. Three of them died of respiratory failure during the immediate postoperative period. Twelve dogs were studied between postoperative day 1 and day 7 to assess the viability of the graft, and a second group of 12 dogs was studied between day 7 and 60 after placement of the graft.

Endoscopic examination, after removal of the endoprosthesis (between 10 and 14 postoperative days), showed the walls of the graft to be rigid; therefore, the new airway did not collapse. Fourteen days after operation, no macroscopic abnormalities in the transitional tracheointestinal areas were observed. The stitches were visible in the lumen of the new airway. The inside surface of the graft was pink, and no breaks were evident. The diameter of the neotrachea showed a reduction of about

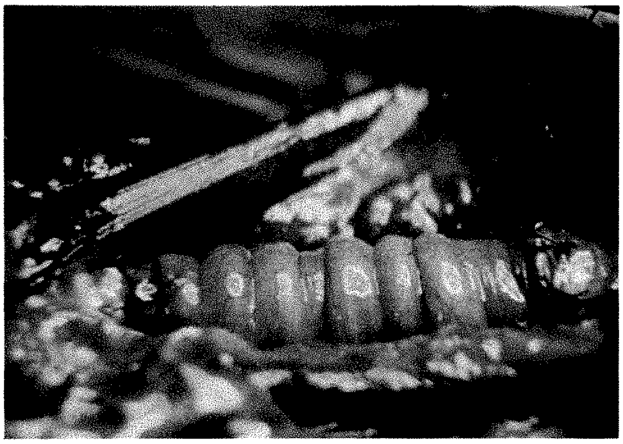

Fig 2. *The intestinal loop has been transferred to the neck, and tracheal interposition has been completed. The tracheointestinal anastomoses have been performed, and the viability of the graft was assessed by peristaltic contraction.*

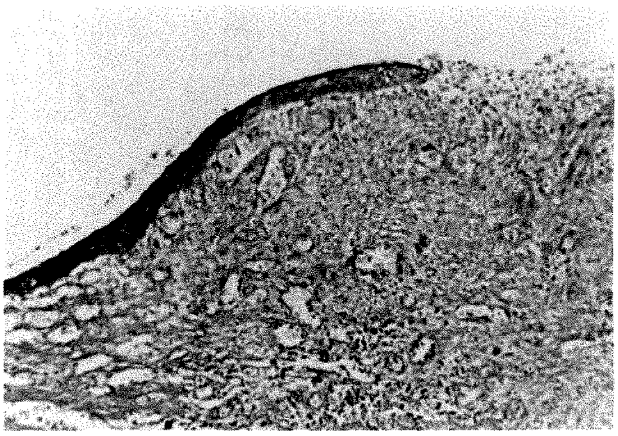

Fig 3. *Granulation tissue replacing the mucous membrane and progressively covered by squamous epithelium originating in the adjacent tracheal epithelium. (Hematoxylin and eosin, ×40 before 54% reduction.)*

30%, which no doubt was related to the size of the stent (tracheal diameter, 1.9 cm; intestinal diameter, 1.4 cm).

No mucous plugs were observed, and the lumens of the new airway, the upper and lower tracheal segments, and the main bronchi were perfectly permeable. Animals were killed at a mean time of 21 days up to a maximal survival of 60 days; thus, long-term results are not available.

Microscopic study showed well-preserved tracheal mucosa with moderate inflammatory infiltration of a chronic type. A local increase in mucous glandular component was observed. The epithelium of the tracheal mucosa had been replaced by nonkeratinizing metaplastic polystratified squamous epithelium. The tracheointestinal anastomosis showed a squamous metaplasia in the tracheal zone that developed into a squamous epithelium covering the intestinal portion next to the anastomosis.

The submucosa, muscular, and serous layers of the graft were well preserved. The mucous membrane had been replaced by well-vascularized granulating tissue, and in the peripheral areas close to the tracheal anastomosis a partial covering by squamous epithelium, probably originating in the adjacent tracheal epithelium, was evident (Fig 3). From 3 weeks on, covering of up to 20% of the surface of the graft was observed.

A study of the mesentery and surrounding cervical tissue showed adherence of the mesentery of the intestinal vascular bundle to the adjacent tissues by peripheral connective tissue, forming weak connections through collagen fibers and fibroblasts associated with small vessels. With regard to the intestine, the areas of maximum adherence were those in which connective tissue surrounded the bundles of skeletal muscles of the neck, which probably accounted for fixation of the graft and prevention of its collapse (Figs 4–6). These muscular fibers underwent degenerative changes in the form of loss of muscular fibers and increase in the connective component together with regenerative changes with irregular fibers containing multiple centrally located nuclei.

Comment

The reconstruction of extensive tracheal defects (more than 50% of the total length of the trachea) requires use of grafting techniques. At present, no ideal technique exists, although the molded Silastic prosthesis as devised by Neville and colleagues [11, 12] is the most commonly used.

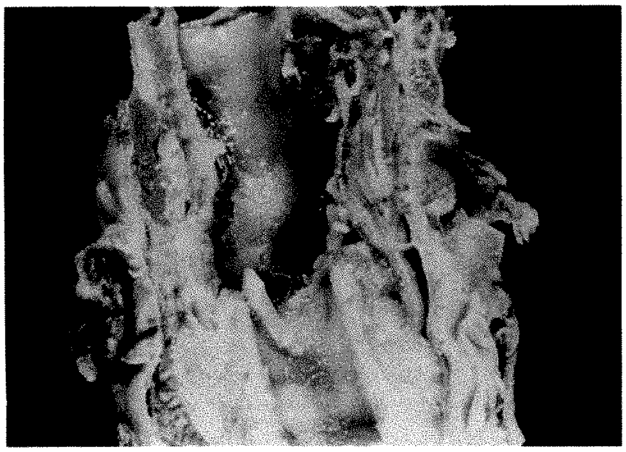

Fig 4. Macroscopic appearance of the transitional tracheal/graft zone 21 days after operation. The caliber of the intestine is adequate.

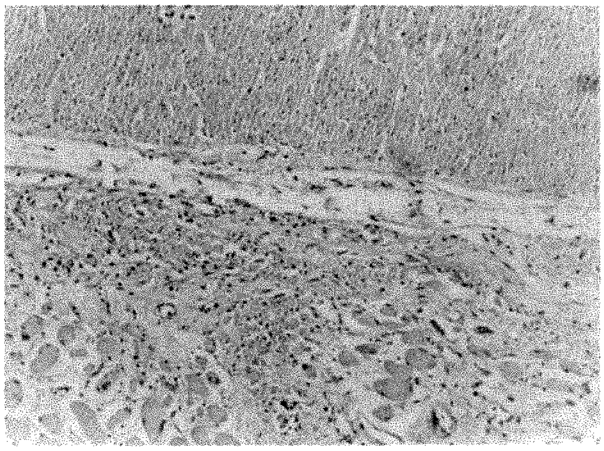

Fig 5. Photomicrograph (hematoxylin and eosin, ×40 before 54% reduction) 10 days after operation: connective tissue adherence between the intestinal loop and the muscular tissue of the neck is apparent.

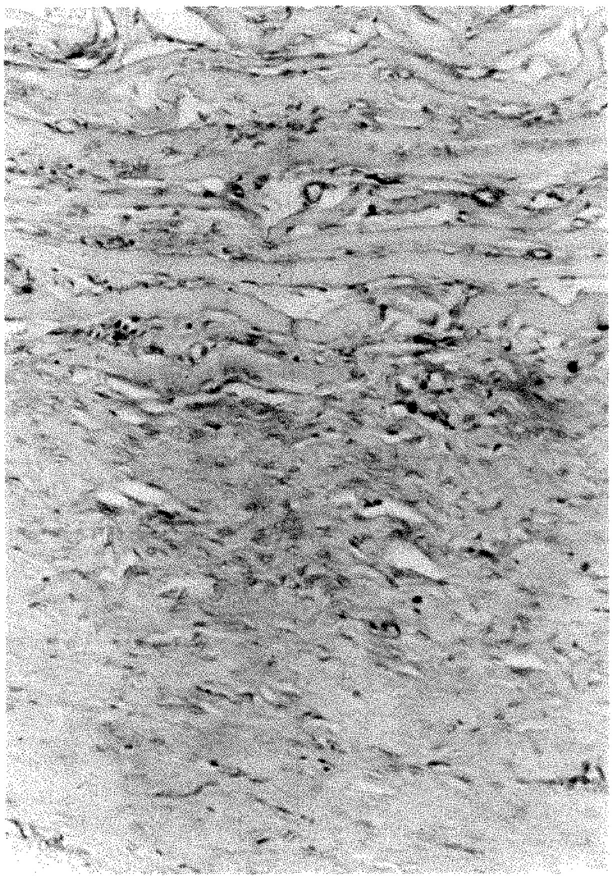

Fig 6. Photomicrograph (hematoxylin and eosin, ×100 before 35% reduction) of an advanced stage, with dense fibrous tissue between the external muscular layer of the graft and the muscular tissue of the neck.

By using a segment of small bowel, the required length and rigidity in the graft can be obtained. The absence of collapse in the new airway after removal of the endoprosthesis was already apparent during the second week after operation and may be attributed to the development of connective tissue attaching the muscular structures surrounding the graft to the serous layer of the same.

Histological examination of the graft 2 weeks after replacement of the trachea showed preservation of the submucosa, muscular, and serous layers. The most important changes were the replacement of the mucous membrane and, in the areas close to the tracheal anastomoses, the development of squamous epithelium originating in the surrounding tracheal epithelium and covering the granulation tissue.

The silicone stent, which initially supported the new airway, was removed during the second week after operation. It was well tolerated and served its purpose in maintaining the airway open until the grafted small bowel section became attached to the neighboring muscles. No hypersecretion or mucous formation obstructing the animal's air passages was observed. Both endoscopic and macroscopic examinations confirmed the permeability of the tracheointestinal anastomoses.

The absence of malacia was confirmed by fiberoptic examination immediately after operation and by the clinical status of the animals from day 14 until the moment the last animals were killed 60 days after operation.

The complications that arose in our series were largely due to necrosis of the graft as a result of vascular thrombosis and the leakage of microsuture. We believe that the results obtained offer new perspectives in tracheal reconstruction techniques for lesions requiring removal of more than 6 cm of tracheal length [14].

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Operative Findings After Percutaneous Mitral Dilation

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Ten patients were operated on within one hour to 3 months after percutaneous mitral dilation. Operative analysis of the lesions demonstrated hemopericardium (n = 3), acute mitral insufficiency (n = 7), left atrial thrombus (n = 2), and atrial septal defect (n = 3). A conservative treatment of the mitral valve lesion was performed in 6 patients. All patients had uneventful postoperative courses. The complications presented in this series were either due to technical error or occurred in patients with relative contraindications to percutaneous mitral dilation (calcified commissures).

(Ann Thorac Surg 1990;49:959–63)

Percutaneous mitral dilation (PMD) is an efficacious technique in the treatment of certain types of mitral stenoses [1–6]. The morbidity and mortality of PMD are very low [7, 8]. Aside from perforation of a chamber resulting in hemopericardium, the complications of PMD are usually well tolerated and do not require urgent operative intervention [7, 8].

This report examines a series of patients operated on after failed or complicated PMD, most of whom required surgical intervention within 12 hours because of cardiac tamponade or severe acute mitral regurgitation.

Material and Methods

Since 1986, 10 patients, aged 20 to 54 years, have been operated on after PMD. Urgent surgical intervention (less than 12 hours after PMD) was required in 7 patients. All patients were seen with either pure mitral stenosis or mitral stenosis associated with minimal mitral insufficiency, and 9 of the 10 were in normal sinus rhythm before PMD.

Percutaneous mitral dilation was accomplished by a transseptal approach to the mitral orifice after femoral vein catheterization. Two balloons (one conventional and one trefoil) were inflated simultaneously, and the results were evaluated by echocardiography after each inflation.

Results

Table 1 shows a clinical summary of the results.

Hemopericardium (n = 3)

Two patients experienced thoracic pain accompanied by hypotension that responded to volume resuscitation during attempts at transseptal puncture. Urgent sternotomy (three to six hours) was performed after echocardiography had shown the existence of increased pericardial fluid. In both patients, right atrial perforation was found and subsequently repaired, and commissurotomy of the stenosed mitral valve was performed in the same operative procedure.

In a separate patient, inflation of the commissurotomy balloon was followed within several minutes by severe hemodynamic collapse with clinical signs of cardiac tamponade. The patient was immediately taken to the operating room. Findings consisted of serious hemopericardium due to a perforation of the left ventricular apex, which was surrounded by a large intramyocardial hematoma. The hemopericardium was evacuated and the ventricular tear was sutured using Teflon strips. The mitral stenosis was surgically treated at a later date.

Acute Mitral Insufficiency (n = 5)

Five patients were seen with clinically acute mitral insufficiency after PMD (Fig 1). In 2 patients, acute mitral insufficiency produced an unstable hemodynamic state with massive pulmonary edema (V wave of 50 to 70 mm Hg). Surgical intervention followed within two to three hours. The lesions responsible for the mitral regurgitation were diagnosed by transthoracic echocardiography immediately after PMD, and were confirmed by operative analysis of the valve.

Rupture of the anterior papillary muscle occurred in 1 patient, who had a complete rupture of the papillary muscle at its zone of insertion on the left ventricular muscular wall. The commissures were otherwise satisfactorily opened and the subvalvular apparatus was moderately diseased (shortened chordae tendineae). A Carpentier-Edwards mitral bioprosthesis was used for valvular replacement. A tear of the interatrial septum at the site of the fossa ovalis (2 cm in length) was closed and a small mural thrombus adherent to the left atrial wall was removed.

Tear of the anterior leaflet occurred in another patient, who had asymmetrical mitral stenosis with mild posterior commissural fusion and an extremely involved and calcified anterior commissure (up to the midpoint of the mitral orifice). When the left atrium was opened, a star-shaped

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Table 1. Operative Findings After Percutaneous Mitral Dilation (n = 10)

Patient No.	Timpe Lapse	Operative Findings	Surgical Treatment
1	<1 h	Perforation of LV	Repair of bleeding site
2	3 h	Perforation of RA	Repair of bleeding site + MC
3	6 h	Perforation of RA	Repair of bleeding site + MC
4	2 h	AMI + LAT + ASD	MVR + closure of ASD
5	3 h	AMI	MVR
6	8 h	AMI	Mitral valve repair
7	12 h	AMI	Mitral valve repair
8	13 days	AMI + LAT	MVR
9	90 days	AMI + ASD	MVR + closure of ASD
10	5 days	AMI + ASD	Mitral valve repair + closure of ASD

AMI = acute mitral insufficiency; ASD = atrial septal defect; LAT = left atrial thrombus; LV = left ventricle; MC = mitral commissurotomy; MVR = mitral valve replacement; RA = right atrium.

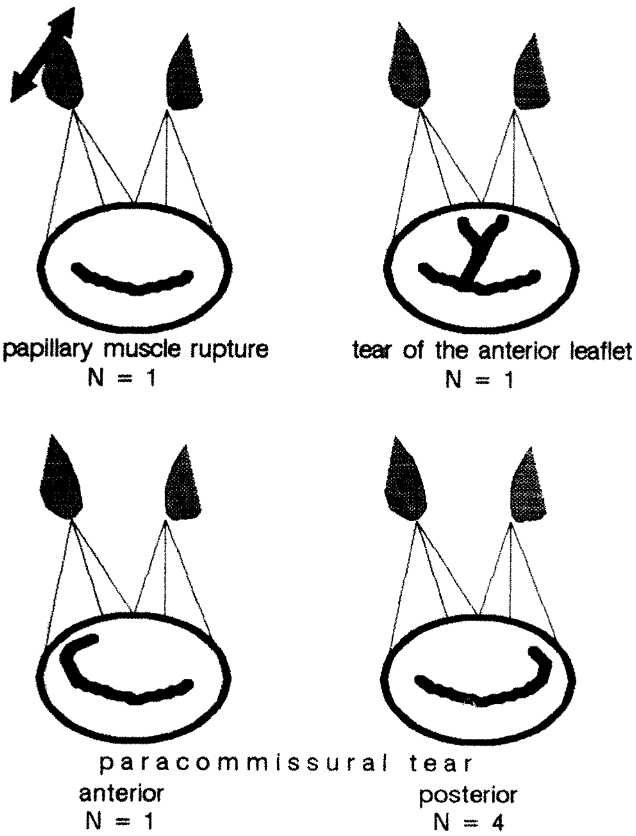

Fig 1. Mechanisms of acute mitral insufficiency after percutaneous mitral dilation: papillary muscle rupture (n = 1), anterior leaflet tear (n = 1), and paracommissural tear (n = 5).

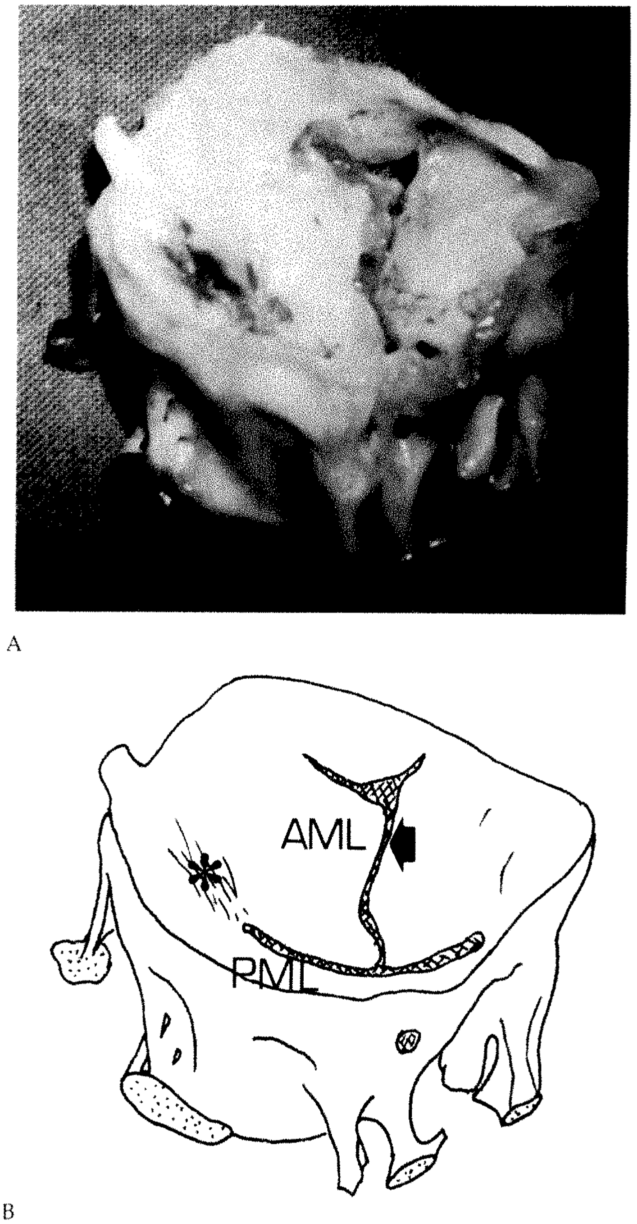

Fig 2. Star-shaped tear of the medial portion of the anterior mitral leaflet (AML) *(arrow). Note the calcified anterior commissure (*) with papillary muscle-commissure fusion.* (PML = *posterior mitral leaflet.)*

tear of the anterior leaflet was found (Fig 2). An attempt at conservative repair was not possible because of the severe involvement of the subvalvular apparatus (fusion of the papillary muscle and anterior commissure), and the valve was replaced with a St. Jude prosthesis.

The remaining 3 cases of acute mitral insufficiency, which was initially poorly tolerated (pulmonary edema), were eventually able to be controlled by medical therapy. In 2 of these patients, rapid operative treatment (eight to 12 hours) was necessary in light of massive mitral regurgitation. The responsible lesions were paracommissural valvular tear causing a localized disinsertion of either the anterior (n = 1) or the posterior (n = 1) leaflet (Fig 1). In both patients, there was no serious involvement of the subvalvular apparatus and the commissures were satisfactorily opened. Surgical reconstruction of the mitral valve was accomplished by reparative suturing of the torn leaflet and Carpentier ring annuloplasty (No. 30).

In 1 case, a 43-year-old woman with predominant mitral stenosis was seen with a massively enlarged left atrium (cardiothoracic index = 0.91) (Fig 3A). Transesophageal echocardiography did not show the presence of a left atrial thrombus, and PMD was performed. A satisfactory commissurotomy was accomplished; however, severe mitral insufficiency ensued and was followed by serious pulmonary edema, which resolved rapidly. A left atrial thrombus was found by magnetic resonance imaging (Fig 3B), and the patient was referred to operation 2 weeks later.

Operative analysis of the lesion demonstrated a massively enlarged left atrium with an old thrombus (4 cm in diameter) protruding from the floor of the atrium approximately 3 cm from the mitral valve orifice, fibrous involvement of the mitral valve apparatus with a posterior paracommissural tear, and tricuspid insufficiency secondary to annular dilatation. These lesions were treated by (1) removal of the thrombus, (2) left atrial plication, (3) Starr-Edwards mitral valve replacement, and (4) Carpentier ring tricuspid annuloplasty.

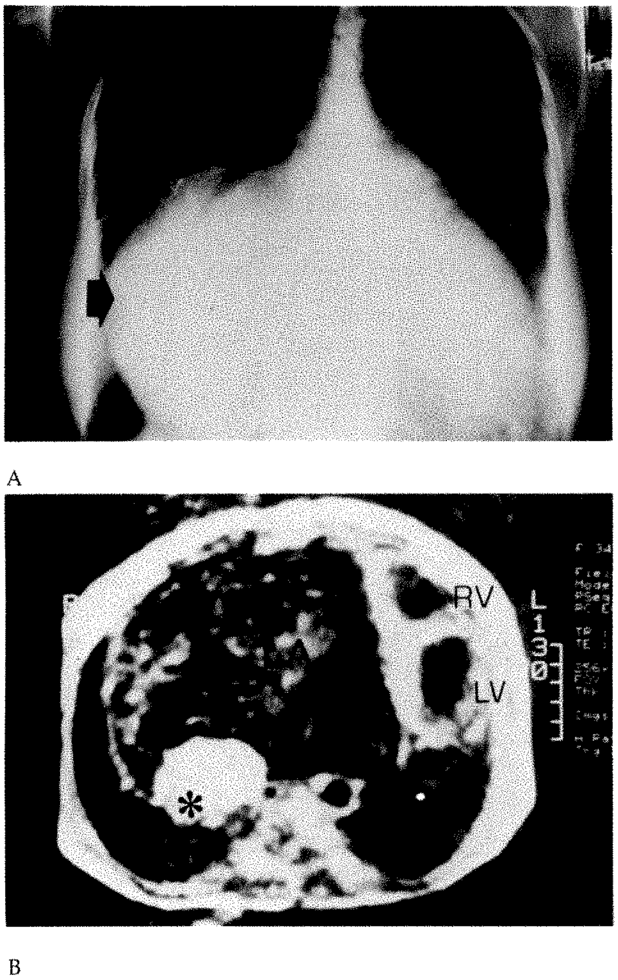

Fig 3. (A) Roentgenogram showing cardiomegaly due to massively enlarged left atrium (arrow). *(B) Magnetic resonance imaging (transverse cut) showing massive enlargement of the left atrium* (LA) *with thrombus (*) seen just to the right of the vertebral column.* (RV = *right ventricle;* LV = *left ventricle.)*

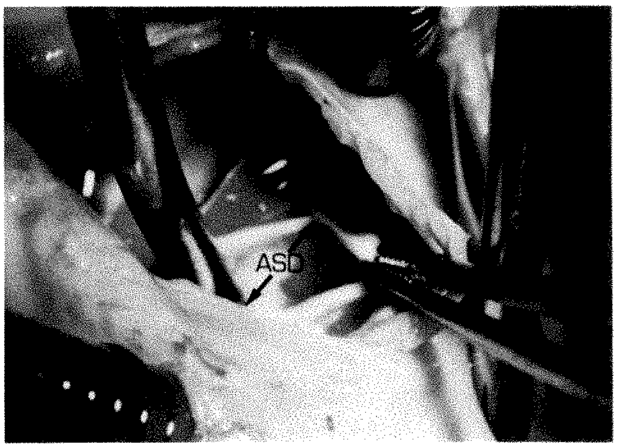

Fig 4. Opening of the right atrium: presence of an atrial septal defect (ASD).

Left-to-Right Shunt Associated With Mitral Insufficiency (n = 2)

In a 43-year-old patient with pure mitral stenosis and pulmonary hypertension (mean pulmonary arterial pressure = 30 mm Hg), a transesophageal echocardiography after PMD demonstrated good commissural opening (from 0.8 cm^2 to 1.8 cm^2), moderate mitral insufficiency of new onset, and the existence of a 2.3:1 left-to-right shunt secondary to an intraatrial communication.

Because of the progressive worsening of the patient's clinical status over the ensuing 3 months, she was referred to operation. Through a right atriotomy, a 3.5-cm endothelialized vertical tear at the site of the fossa ovalis (Fig 4) was demonstrated. Tricuspid insufficiency due to annular dilatation was also noted. The mitral valve was torn at the posterior commissural area, and there was extensive calcification of the anterior commissure. Surgical treatment was comprised of primary closure of the intraatrial communication, mitral valvular replacement using a St. Jude prosthesis, and tricuspid annuloplasty with a No. 34 Carpentier ring.

In a 34-year-old woman, the PMD procedure was followed by severe mitral regurgitation, which was surgically treated five days later. In addition, transesophageal echocardiography revealed the existence of consistent left-to-right shunt (2.2:1). A paracommissural tear of the posterior commissure was repaired by resuturing, and a satisfactory open commissurotomy was realized. Primary closure of a small atrial septal defect (1.5 cm) was simultaneously performed.

There was no operative mortality in this series. All patients had uneventful postoperative courses and exhibited good functional results.

Comment

Since the first reports by Inoue and associates [1] in 1984, indications for PMD have had a tendency to be liberalized [2–6]. According to the cardiology literature, complications from this technique are rare, and the mortality is minimal [7, 8]. However, there are two post-PMD situations that require urgent surgical intervention: cardiac tamponade and severe acute mitral insufficiency.

Tamponade can be secondary to a puncture of the right atrium during attempts at transseptal catheterization (2 patients in this series). These events are of variable gravity, but can be well tolerated. Echocardiography will confirm the existence of pericardial fluid that had been absent before PMD. In these circumstances, it is logical to simultaneously treat the hemopericardium as well as the mitral valve pathology.

Hemopericardium secondary to perforation of the left ventricle with severe hemodynamic compromise occurred in 1 patient. This was probably secondary to balloon slippage at the time of inflation, causing left ventricular apical injury and an intramural hematoma that subsequently exteriorized into the free pericardium. This complication necessitated immediate control of the ventricular tear.

Acute mitral insufficiency secondary to PMD can have very different clinical presentations, depending on the mechanical lesion incurred. The onset of massive pulmonary edema associated with serious hemodynamic instability refractory to medical therapy frequently signifies severe trauma to the mitral apparatus. Transthoracic echocardiography allows precise identification of the lesion.

This complication was observed in 2 patients, who required surgical intervention within two to four hours. In 1 patient, papillary muscle rupture at its ventricular insertion was probably the consequence of balloon inflation between the subvalvular apparatus and the ventricular wall [9]. Routine use of a floating balloon catheter before insertion of the PMD balloon, to assure correct positioning of it within the mitral orifice, should prevent this complication. In the second patient, an extensive tear of the anterior leaflet was present. The leaflet injury was located at its midportion because of substantial asymmetry of the mitral stenosis (anterior commissural calcification and fusion to the midportion of the mitral orifice with a normal posterior commissure). In retrospect, this may have been a contraindication to PMD.

Acute mitral insufficiency after PMD can also manifest itself by initial pulmonary edema, which favorably responds to medical treatment. In 2 patients, rapid operative intervention was carried out because of massive mitral insufficiency. Each time, a disinsertion of a mitral leaflet secondary to a paraannular tear at the site of a commissure was found. These lesions were treated by simple resuturing of the leaflet and Carpentier ring annuloplasty. Interestingly, in both cases the diameter of the mitral annulus was relatively small (No. 30 Carpentier ring). Radial twisting of the balloon during inflation while in the mitral orifice was the probable mode of injury.

The possibility of a conservative reconstructive repair of the mitral valve after PMD does not seem to be related to the extent of the valvular tear, which is usually easily suturable, but is related more importantly to the degree of pathological involvement of the valvular and subvalvular apparatus (ie, calcification of the commissures), which may necessitate valve replacement.

In spite of the usefulness of transesophageal echocardiography in the diagnosis of intraatrial thrombi [10], this technique was inaccurate in 2 cases in this series (1 case involved a small adherent thrombus, and the other, a large thrombus protruding into a massively enlarged left atrium). Fortunately, no embolic complications occurred after PMD.

The existence of associated lesions that are primarily amenable to surgical therapy (left atrial ectasia, severe tricuspid insufficiency, thrombus of the left atrium) should provoke consideration of offering surgical treatment of mitral stenosis without first attempting PMD.

The onset of a left-to-right shunt after PMD is not rare [11], although it is usually a modest shunt without clinical consequence (shunt fraction 1.3:1 to 1.8:1) [12]. The intraatrial communications after PMD reported in this series are exceptionally large (2.3:1). This shunt was probably poorly tolerated because of the onset of mitral insufficiency secondary to PMD, as well as the preexistence of pulmonary arterial hypertension and tricuspid insufficiency. As in other reports in the literature [12, 13], these complications may be the consequence of premature PMD catheter withdrawal before complete balloon deflation.

The patients operated on in this series were referred from different cardiology departments who do not refer all their complications to us; consequently, it seems difficult to establish the exact incidence of complicated PMD. However, the complications of PMD reported here are rare according to the cardiology literature [7, 8], and no mortality or eventual serious sequelae related to PMD were observed in our series. The complications were either due to technical error or to PMD in patients with relative contraindications (ie, calcified commissures).

We do not believe that a permanent surgical standby is mandatory for PMD procedures performed by experienced cardiology centers; when complications do occur, there is usually ample time to allow mobilization of the cardiac surgical team. In rare instances, readily available on-site circulatory support equipment in the catheterization laboratory could be advantageously used to assure transfer of the patient to the operating room in a satisfactory hemodynamic condition.

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Case-Matched Comparison of Mitral Valve Replacement and Repair

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Carpentier's techniques of prosthetic ring mitral valve repair for mitral regurgitation offer the potential for immediate and long-term improvement in valve function without the necessity of replacing the native valve with a prosthesis. A consecutive, case-matched series of 65 patients with prosthetic ring mitral valve repair was compared with 65 patients undergoing mitral valve replacement for mitral regurgitation. The aortic cross-clamp time was 57 ± 33 minutes in the repair operations and 41 ± 25 minutes in the replacement operations ($p = 0.003$). The cardiopulmonary bypass time was 154 ± 44 minutes in the repair operations and 113 ± 41 minutes in the replacement operations ($p = 0.0001$). There were no myocardial infarctions in the hospital in either group. Hospital death was noted in 1.5% of repairs and 4.6% of replacements (p = not significant). Survival at 4 years was 0.84 for repairs and 0.82 for replacements (p = not significant). Freedom from reoperation to replace the mitral valve at 4 years was 62 of 65 patients in the repair group and 64 of 65 patients in the replacement group (p = not significant). In-hospital and midterm results in a closely matched population show that mitral valve repair yields results comparable with those of replacement despite a more difficult procedure. The benefits of maintaining the native valve with chordal and papillary muscle structure intact and avoidance of prosthetic valve implantation may then become apparent with longer follow-up.

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Mitral valve repair to correct mitral regurgitation using the techniques of Carpentier has shown an advantage in the treatment of mitral valve disease [1–4]. In comparison with replacement, mitral valve repair offers a reduced incidence of thromboembolism and the necessity of anticoagulation related to use of mechanical valves [4]. There is also a potential for longer functional durability with the patient's own repaired valve than has been achieved with a bioprosthetic device [5–9]. In addition, maintenance of the mitral valve and its chordal-papillary muscle mechanism has been documented in both clinical and laboratory reports to play an important role in maintaining ventricular function [10–12]. Preservation of the valve apparatus is of particular importance in supporting postoperative ventricular function in patients with chronic mitral valve regurgitation. By using mitral valve repair techniques instead of replacement to correct mitral regurgitation, preservation of this intrinsic support role is maximized.

Although extensive literature exists on both mechanical and bioprosthetic valves in the mitral position and some literature exists on the use of mitral repair, there has been limited direct comparison between these two modes of therapy [5–9, 13, 14]. Thus, the apparent superiority of mitral repair cannot be justified in any patient. Because intraoperative evaluation is ultimately necessary to determine whether a given patient is a suitable candidate for valve repair, it is unlikely that a randomized trial could ever be mounted. In addition, surgeons trained in repair techniques may prefer to perform repair if the valve anatomy permits a relatively straightforward repair. This would result in the randomized patients being limited to only those with more complex regurgitant lesions. Thus, we compared patients undergoing prosthetic ring mitral valve repair using the techniques of Carpentier with an age- and sex-matched group of patients undergoing mitral valve replacement.

Material and Methods

The study patients comprised 65 consecutive patients, elective and emergent, undergoing prosthetic ring mitral valve repair for mitral regurgitation between November 1, 1984, and December 31, 1988, the initial experience with mitral valve repair in this hospital. Of these patients, 20 had associated coronary artery bypass grafting. Patients who had had other cardiac or aortic valvular surgical procedures were excluded. These patients were matched with control patients undergoing mitral valve replacement. The control patients were obtained by computer-matching algorithms from our database containing 637 patients undergoing first mitral valve replacement between 1979 and 1988. The mean difference in time for the matched pairs was 1.2 ± 2.7 years. The control patients all had operation within 5 years of the study patients, were within 6 years of the age of the study patients at the time of operation, were of the same sex, all underwent elective operations, and had the same number (or within 1) of coronary bypass grafts placed.

In 13 additional patients, we hoped to correct mitral regurgitation by repair techniques. In 9 of these, the

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repair effort was started but abandoned and valve replacement was performed when anatomical conditions were found that were believed to preclude a satisfactory repair. These included extensive fibroelastic tissue deficiency (3 patients), unremedial rheumatic scarring (4), and extensive endocardial necrosis from acute myocardial infarction (2). In 4 others, prosthetic ring mitral repair was completed but intraoperative evaluation determined that unacceptable regurgitation remained. All 4 patients had extensive fibroelastic tissue deficiency not amenable to further repair measures, and valve replacement was performed. Nine of the 13 did well. Four hospital deaths occurred owing to factors of the patients' disease and were neither related to nor caused by the repair effort. None of these 13 patients were included in either of the matched populations studied in this report.

The causes of mitral regurgitation in the repair group were as follows: rheumatic disease, 8%; ischemic disease, 8%; degenerative tissue disease, 78%; and endocarditis, 6%. This pattern is similar to that of the replacement group.

Prosthetic ring mitral valve repair based on the Carpentier techniques has been previously defined [1, 15]. Standard cardiopulmonary bypass techniques for intracardiac valve operations were used in all patients. Myocardial protection was achieved with systemic hypothermia, ischemic arrest with cold potassium cardioplegia, and topical hypothermia or intermittent aortic occlusion in all patients. Valve replacement used eight mechanical valves and 57 porcine bioprostheses, of which nine were Hancock valves and 48 were Carpentier-Edwards valves. Mitral repairs included a series of techniques including quadrangular resection of areas of prolapsed leaflet, trench, sliding and cylindrical chordoplasty, and placement of annuloplasty ring [1, 15]. Implantation of a prosthetic annuloplasty ring was performed in all 65 patients having repair. In all patients, however, at least one additional repair technique was required to correct the regurgitation. Complex repairs with multiple techniques were frequent owing to the high incidence of degenerative tissue disease. Specific repair techniques were used as follows: leaflet resection in 54 patients, chordoplasty in 33, marginal transposition in 2, and prosthetic annular ring implantation in 65.

Intraoperative evaluation of the adequacy of valve repair was made by direct visual inspection of leaflet coaptation by observing any regurgitation when the ventricular chamber was filled. The ventricle was filled by the bulb-flush technique [1] or by production of temporary aortic valve insufficiency. Left atrial pressure monitoring was used initially in a search for regurgitation "V waves" after separation from cardiopulmonary bypass in all repaired valves. Intraoperative transesophageal Doppler echocardiography became the standard method of repair evaluation once it became available. Repair status was evaluated clinically and by transthoracic color-flow Doppler echocardiography. Long-term anticoagulation with warfarin was used in all mechanical valves but only in the presence of chronic atrial fibrillation with a large left atrium in patients who received a bioprosthetic valve or a valve repair.

A perioperative myocardial infarction was diagnosed by the appearance of new Q waves or loss of anterior R waves on the electrocardiogram, or by an increase in the level of creatine kinase MB isoenzymes. A neurological event was defined as new focal neurological findings that were either permanent or that resolved. Operation was considered emergent at the discretion of the surgeon, generally for unstable symptoms of angina or heart failure. Follow-up mortality, valve status, history of endocarditis, and thromboembolism were determined by telephone survey.

All data were collected on standardized forms and entered into a computerized database. Data were retrieved by a specialized query language and were analyzed with the statistical package BMDP (BMDP Corp, Los Angeles, CA). All data are displayed as mean ± standard deviation. Categorical data were compared by χ^2 and continuous data by unpaired test. Survival was determined by the Kaplan-Meier method [16]. Survival curves were compared by the Mantel-Cox method [17].

Results

The demographics and operative characteristics of the patients are shown in Table 1. No difference was noted in age, sex, ejection fraction, number of grafts placed, or use of the internal mammary artery in the two groups. Severe mitral regurgitation was noted more frequently in the mitral repair group. Most of the patients required no coronary bypass grafts, but 20 in both groups had coronary artery bypass grafts placed (mean, 1.5 grafts per patient). The aortic cross-clamp time and cardiopulmonary bypass time were prolonged significantly in patients undergoing valve repair.

The complications and postoperative courses are shown in Table 2. Use of the intraaortic balloon pump, incidence of myocardial infarction, and wound infections did not differ between the groups. The incidence of reexploration for bleeding was higher in the group with mitral valve replacement. There was a trend to more neurological events in the patients with mitral valve repair. Each of the six neurological events in the patients with repair was reviewed. These were related in 4 patients to prior stroke, a clinical factor not present in the matched replacement population. There was no significant difference in the incidence of pericarditis, length of in-hospital stay, or in-hospital mortality. The incidence of supraventricular and ventricular arrhythmias was higher in patients with mitral replacement.

In-hospital postoperative evaluation of patients undergoing mitral repair by transthoracic Doppler echocardiography showed marked improvement in the mitral valve function of all evaluated patients. No regurgitation was noted in 28, whereas trace or minimal regurgitation was noted in 10. All were clinically improved. Later follow-up (6 weeks to 2 years) Doppler echocardiographic evaluation is available for 42 patients who had valve repair. Twenty-six of these patients showed no evidence of mitral regurgitation, 4 had trace or minimal regurgitation, and 10 were believed to have mild to moderate regurgitation by this sensitive evaluation method. Severe regurgitation

Table 1. Demographics and Operative Characteristics

Characteristic	Repair (n = 65)	Replacement (n = 65)	p Value
Age (yr)			
Mean	61 ± 12	60 ± 12	NS
Range	27–81	27–78	. . .
Male sex	45 (69%)	45 (69%)	NS
Ejection fraction	0.57 ± 0.11 (n = 25)	0.57 ± 0.13 (n = 40)	NS
Severe mitral regurgitation	24/28 (85.7%)	28/46 (60.9%)	0.02
No. of coronary grafts			
0	45 (69%)	47 (72%)	NS
1	10 (15%)	9 (14%)	. . .
2	6 (9%)	2 (3%)	. . .
3 or more	4 (6%)	7 (10%)	. . .
Internal mammary artery graft	6 (9.2)	3 (4.6)	NS
Aortic cross-clamp time (min)	57 ± 33	41 ± 25	0.003
Cardiopulmonary bypass time (min)	154 ± 44	113 ± 41	0.0001

NS = not significant.

developed in 2 patients 1 month and 6 months postoperatively and required reoperation (discussed later in this section). Systolic anterior motion of the anterior leaflet producing compromise of the ventricular outflow tract was not observed in any patient who had repair despite a careful search after this malady had been reported [13, 18].

Two-year follow-up endpoints are shown in Table 3. There was no significant difference in the incidence of valve failure requiring mitral replacement. Two valves were replaced in the repair group within 2 years of operation, one at 1 month and one at 6 months postoperatively for recurrent mitral regurgitation; both patients had severe fibroelastic deficiency tissue disorder. In 1 patient in the repair group mitral stenosis developed owing to unexplained leaflet fibrosis at 2 years postoperatively and required mitral replacement. One patient in the replacement group required repeat replacement 2 months after operation. There were no late bleeding episodes in the repair group and two episodes in the replacement group at 11 and 23 months. This difference was not significant. There were no cases of endocarditis in the repair group and 1 case in the replacement group at 2 months. This difference was not significant. Late thromboembolism was noted in only 1 patient in the repair group and in none in the replacement group (p = not significant). Four-year survival is shown in Figure 1. Although the survival for patients who had repair was slightly higher throughout the 4-year period, the difference was not statistically different.

Comment

We compared matched patients who had mitral valve replacement with patients who had prosthetic ring mitral repair. A case-matching scheme was used instead of a randomized study. There was remarkably little difference between the groups. Patients who underwent repair had a longer cross-clamp time and bypass time. This was expected, as the repair procedure is more complicated and time-consuming to perform. The incidence of reexploration for bleeding and of arrhythmias was higher in the replacement group. There is no clear explanation for these trends. There was a trend toward more neurological events in the repair group. The repair group, but not the

Table 2. Complications and Postoperative Characteristics

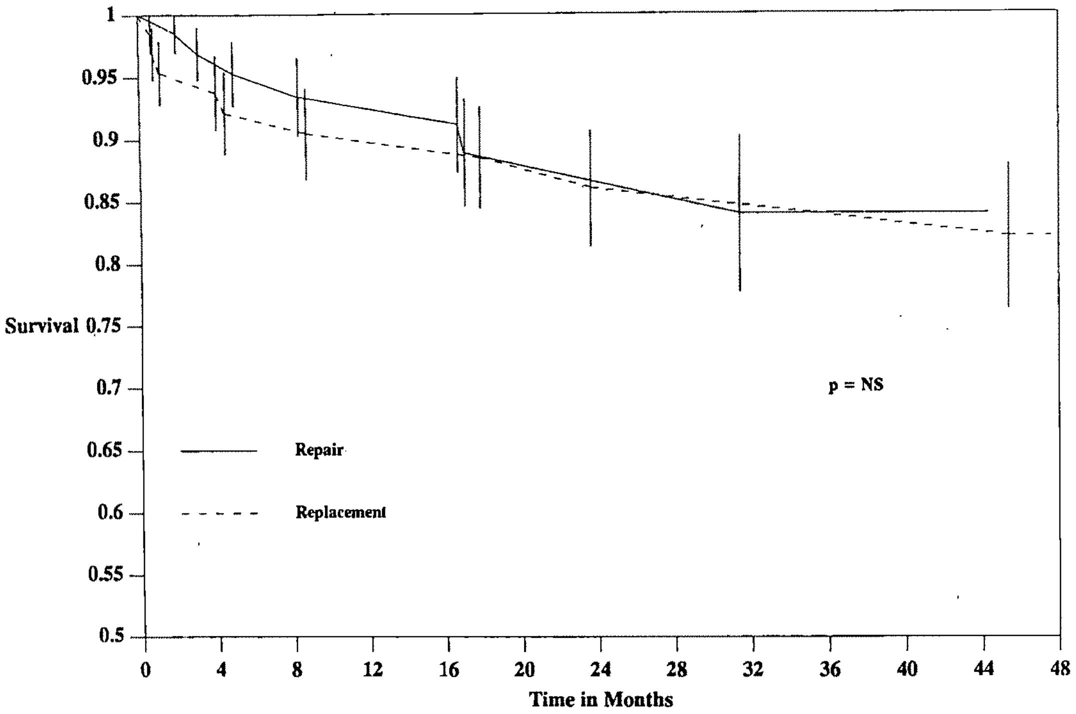
Characteristic	Repair (n = 65)	Replacement (n = 65)	p Value
Intraaortic balloon	3 (4.6%)	6 (9.2%)	NS
Postoperative myocardial infarction	0 (0%)	0 (0%)	NS
Reexploration for bleeding	0 (0%)	4 (6.2%)	0.04
Wound infection	1 (1.5%)	2 (3.1%)	NS
Neurological event	6 (9.2%)	2 (3.1%)	NS
Pericarditis	3 (4.6%)	9 (13.8%)	0.07
Postoperative arrhythmias	24 (37%)	38 (59%)	0.01
Hospital stay (days)	12 ± 12	15 ± 16	NS
Hospital death	2 (1.5%)	3 (4.6%)	NS

NS = not significant.

Table 3. Two-Year Acturial Follow-up Results

Result	Repair (n = 65)	Replacement (n = 65)	p Value
Survival	0.89	0.86	NS
Freedom from valve replacement	0.96	0.98	NS
Freedom from bleeding episodes	1.00	0.98	NS
Freedom from endocarditis	1.00	0.98	NS

NS = not significant.



Repair													
n Remaining	65	63	50	45	43	30	24	19	17	12	6	1	
Survival	1	.97	.95	.93	.93	.89	.89	.89	.84	.84	.84	.84	
Deaths	0	2	3	4	4	6	6	6	7	7	7	7	
Replacement													
n Remaining	65	58	55	51	48	41	35	34	29	26	25	22	19
Survival	1	.94	.92	.90	.90	.88	.86	.86	.86	.86	.86	.86	.82
Deaths	0	4	5	6	6	7	8	8	8	8	8	8	9

Fig 1. Four-year survival curves for mitral repair versus replacement.

replacement group, had a high incidence of prior strokes (4 patients). This preexisting clinical descriptor appeared to have a greater influence on the higher incidence of perioperative stroke in patients having valve repair than did factors related to the valve repair process itself. In 4 patients, the perioperative neurological deficits were transient, but 2 of the patients in the repair group had a residual neurological deficit. This was the same stroke rate as that observed in the replacement group.

Two reports have compared mitral valve repair with mitral valve replacement. Angell and colleagues [14] reported a 10-year study of 184 patients, 112 of whom had valve repair and 72 of whom had replacement with a porcine bioprosthesis. On the average, their replacement group was 5 years older and had a higher percentage of "end-stage" patients who had valve replacement. This bias was believed to be responsible for a higher operative mortality (18.1%) and higher late mortality rate (10.9% per patient-year) than occurred in their repair group, with rates of 3.6% and 2.9%, respectively. The series of Angell and colleagues [14] made no effort to match patient groups by preoperative clinical, hemodynamic, or demographic descriptors and is therefore less comparable with our current report as a comparison of the two techniques. Nonetheless, its information is of interest. Their study showed an incidence of recurrent severe regurgitation requiring reoperation of 9 of 112 in the repair group, higher than that in our patients. There was also an incidence of "stable, non-progressive, mild regurgitation" of 46.3% of patients who had valve repair; another 31.7% had "trivial" regurgitation. These levels were much higher than those in our series; late follow-up by Doppler echocardiography showed that only 25% had minimal (1+) regurgitation and that 10% had trace regurgitation. Sixty percent of our patients with repaired valves had no regurgitation. The patients in our series with mild residual regurgitation, like those in the study of Angell and colleagues [14], were all hemodynamically and clinically improved and reoperation was not contemplated.

The long-term valve-related problems in patients who underwent mitral valve repair were few in both series. The anticipated hemodynamic advantages believed to be inherent in valve repair could not be demonstrated by the data of Angell and colleagues [14], however. Their conclusion for high-risk patients was to use valve replacement with implantation techniques designed to preserve chordal support from both anterior and posterior leaflets. In our series, several desperately ill patients had valve repair and did well with less postoperative difficulty than was experienced by similar patients who had valve replacement, even with preservation of chordal intrinsic support. Therefore, we do not hesitate to use repair techniques in critically ill patients with poor ventricular function when the repair can be accomplished expeditiously and with expectations of a lasting repair on the basis of tissue status. When a very complex repair is required, or when extensive degenerative tissues are present, we recommend valve implantation with preservation of intrinsic support by incorporating anterior and posterior leaflet margins with their attached chordae into the suture line [11, 12, 19].

Despite a high incidence of reoperation for failure of repaired mitral valves in the series of Angell and colleagues [14], the superior long-term clinical stability and low valve-related morbidity and mortality led them to recommend mitral valve repair as an alternative to valve replacement, particularly for younger patients.

Cohn and co-workers [13] reported comparative, consecutive, concomitant experiences involving 140 patients, 75 having mitral repair and 65 having mitral replacement. Even though the frequency of concomitant coronary bypass grafts was similar to that in our series, the incidence of ischemic causes for the mitral regurgitation in their series was greater, 36% versus 8% in our series. Other clinical and hemodynamic variables were similar among the two series. Cohn and co-workers [13] used both the rigid Carpentier ring and the flexible Duran ring; in 18 patients no ring was used. No statistically significant difference was observed between the early hemodynamic findings in patients in whom the different types of ring annuloplasty were used and patients in whom none was used. They observed similar survival in both groups whether coronary artery bypass grafting was required or not. No incidence of ventricular outflow obstruction resulting from systolic anterior motion was observed in their repair group or in our series.

Although they applied repair techniques readily, Cohn and co-workers [13] advocated valve replacement whenever marked annular calcification or extensive degeneration of multiple chordal elements was present. This philosophy differed from our experience, in which repair was used whenever the tissue elements of the valve indicated that repair was possible, a philosophy to which we continue to adhere.

Although Cohn and co-workers [13] had a higher incidence of early repair failure that required reoperation in their initial experience than we did, the occurrence of mild, clinically insignificant, and nonprogressive late regurgitation was similar. Despite the presence of a soft murmur of insufficiency and documentation of mild or even moderate regurgitation by Doppler echocardiography, clinical improvement was evident in all these patients. The consensus is that these small amounts of mitral insufficiency observed in 25% of patients after mitral repair are preferable to implantation of a prosthetic or bioprosthetic valve with its attendant morbidity [5–8, 12].

Use of intraoperative transesophageal color-flow Doppler echocardiography [20] to assess the status of valve repair immediately after separation from bypass provides an opportunity for the surgeon to correct any persistent insufficiency before leaving the operating room and thus probably could have avoided the high incidence of early reoperation required in 5 patients in the series of Cohn and co-workers [13]. It is far superior and more physiological than visual inspection or flush techniques, which must be used before the atrium is closed and cardiac function restored.

Several series have compared the results of mitral repairs with all mitral replacements for mitral regurgitation or for mitral regurgitation of specific pathological types without efforts to match the two groups clinically. Duran and colleagues [21] reviewed their experience in Spain, where 87 valvoplasties were performed in 85 patients, with an operative mortality of 2.4%. Their patient population was younger than ours, with a mean age of 39 years. Their study population is not comparable with ours because many patients had mitral repair for mitral stenosis and thus cannot be used to determine whether repair or replacement should be used for mitral regurgitation. Orszulak and associates [22] reported a comparative series with repair of mitral regurgitation due to ruptured chordae in 131 patients with a mean age of 57 years. The operative mortality was 6.1%, with a 5-year survival of 92%. In contrast, the operative mortality for mitral valve replacement was 7.5% with a 5-year survival of 72%. The cases were not matched, and the valve replacement patients were older. Yacoub and co-workers [18] operated on 132 patients for mitral valve prolapse. There were 46 replacements and 86 repairs. The mortality was 11% for replacements and 5% for repairs. Their late survival was improved in repairs as well. Oliveira and colleagues [23] studied 183 patients of whom 101 had mitral replacement; mitral repair was attempted in 82 patients and was successfully completed in 71. The 6-year survival was 68% for replacements and 88% for repairs. These studies in nonmatched groups generally showed lower operative mortality and improved long-term survival in patients who had mitral repair. A selection bias in which the more favorable patients and lesions were corrected by repair may explain why repair results were better in each of these series. In contrast, our present study was different in that the patients were individually case-matched by multiple preoperative demographic and clinical descriptors. This may explain the improved results of our patients who underwent valve replacement in contrast to the data on valve replacement in the series of Orszulak and associates [22], Yacoub and co-workers [18], and Oliviera and colleagues [23], whereas our data for patients having valve repair are comparable in our series and in theirs.

Whether mitral repair could be applied to our population as effectively and with the favorable results achieved in the European series was answered by this study. We have shown that the in-hospital and midterm follow-up results of patients with prosthetic ring mitral repair are comparable with those of European studies as well as those of patients who have mitral valve replacement. The incidence of problems related to repair disruption that required reoperation were minimal even in this series, our initial experience. In each case, recurrent mitral regurgitation was related to intrinsic fibroelastic deficiency of the valve tissue rather than failure of technical repair, which does not mean that all patients with mitral regurgitation can or should have valve repair. Clearly this is not the point, even though all patients who underwent valve repair could have had replacement. Furthermore, the favorable comparison was achieved in a population having valve repair that was older than the average age for referral for mitral valve replacement and included several patients having emergency operations.

The advantages and justification for using the more technically intricate procedure of repair for our patients may become stronger after longer periods of follow-up. Perrier and colleagues [4] reported a concomitant series of 400 patients who had mitral operations to correct mitral regurgitation. One hundred patients received prosthetic ring mitral repair or valve replacement with bioprosthetic, Björk-Shiley, or Starr-Edwards valves. The report of Perrier and colleagues [4] emphasized the durability and freedom from late complications with mitral repair in comparison with valve replacement. Because the mitral repair procedure uses living, autogenous tissue, a continued lower rate of thromboembolism and freedom from the morbidity of prosthetic devices and long-term anticoagulation should enhance favorably the further comparison of repair versus replacement as follow-up intervals lengthen.

With this report demonstrating favorable comparison of valve repair with valve replacement in a matched population, and with data now being reported of the limited durability of bioprosthetic valves and the late morbidity and mortality of mechanical valves [11, 12], the durability and low late complication rate of mitral repair as well as its maintenance of the intrinsic ventricular support mechanism warrants application of this technique whenever feasible. The improved long-term results to be expected from mitral valve repair may also justify earlier referral in the patient's course of disease. Thus, the maximum long-term results can be obtained before ventricular deterioration occurs, which would independently limit the long-term benefit of valve operations.

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Isolated Myocardial Perfusion During Arch Repair

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Repair of coarctation of the aorta with severe hypoplasia of the aortic arch or interrupted aortic arch was performed in 5 patients using a modification of the usual technique that consisted of isolated myocardial perfusion during arch repair. The aortic cross-clamp was placed on the ascending aorta distal to the aortic cannula. Cardiopulmonary bypass flow was reduced to about 10% of full flow, achieving a line pressure of 35 to 45 mm Hg to keep the heart perfused and beating during arch repair. Once the aortic arch was repaired, total body perfusion was continued as usual and intracardiac repair was performed. Isolated myocardial perfusion for aortic arch reconstruction reduces myocardial ischemic time.

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Management of coarctation of the aorta with severe hypoplasia of the aortic arch or interrupted aortic arch is a surgical challenge. Recently several groups of investigators [1–5] have presented series of patients treated by simultaneous repair of the arch and intracardiac anomalies. The fragility of these patients, coupled with the potentially long myocardial ischemic time during repair of additional complex intracardiac abnormalities, led to adoption of the technique of isolated myocardial perfusion to reduce myocardial ischemic time.

Technique

An arterial pressure monitoring line is placed in the right radial artery. Median sternotomy is performed and the left thymus is removed to facilitate exposure. The aortic arch and branches are dissected out and the duct and upper descending aorta are exposed (Fig 1A).

Arterial cannulas are inserted in the ascending aorta and the ductus arteriosus or main pulmonary artery using a Y-connector. Snares are placed around the right and left pulmonary arteries to control pulmonary blood flow. A single venous cannula is placed in the right atrial appendage. The arterial cannulas (usually 10F for neonates) must be the same size to ensure that vascular resistance is the only determinant of flow in each of the two separate vascular beds being supplied by these cannulas.

Cardiopulmonary bypass is instituted at the normal flow (150 mL/kg/min) and the patient is cooled down to a nasopharyngeal temperature of 16°C or less and an esophageal temperature of 10° to 12°C. Snares around the right and left pulmonary arteries are snugged down soon after institution of bypass. Perfusion pressure is maintained at about 30 to 45 mm Hg.

When the patient is fully cooled, the ductal cannula is clamped and removed. Flow is reduced to about 10% of calculated flow (line pressure, 35 to 45 mm Hg), and a clamp is placed on the ascending aorta just distal to the aortic cannula. This allows blood to enter the coronary arteries, keeping the heart perfused and beating. Snares are tightened around the head vessels, and the duct is ligated proximally with silk sutures. The descending aorta is cross-clamped as low as possible, and all ductus tissue is excised (Fig 1B). The proximal aortic arch distal to the clamp is opened inferiorly, and the descending aorta is anastomosed end to side with running 7-0 Prolene (Ethicon, Somerville, NJ) suture. After completion of arch repair, vascular clamps on the descending aorta and ascending aorta are removed and the snares on the head vessels are released. Perfusion flow is returned to normal, and intracardiac repair is then performed (Fig 1C).

Results

The results are summarized in Table 1.

Comment

Interrupted aortic arch and coarctation of the aorta with severe hypoplasia of the aortic arch are rare congenital malformations that usually occur with severe associated intracardiac congenital anomalies [6, 7]. The traditional left thoracotomy and repair of the arch is complicated by poor exposure of the proximal arch and the marked difficulty in palliating infants with more complicated lesions such as transposition of the great arteries, truncus arteriosus, aortic stenosis, or subaortic stenosis [8, 9].

In recent years, some surgeons [1–5] have advocated repair of the aortic arch and intracardiac defects simultaneously, and we concur. By using the isolated myocardial perfusion technique, we reduced myocardial ischemic time in our patients by 16 to 41 minutes (mean, 23 minutes). It was limited to 71 to 86 minutes (mean, 78 minutes) in 3 patients who also underwent arterial switch and ventricular septal defect closure. During isolated myocardial perfusion, the heart beats slowly at the low temperature. We conclude that isolated myocardial perfusion may offer an advantage during aortic arch reconstruction without impairing the operative field.

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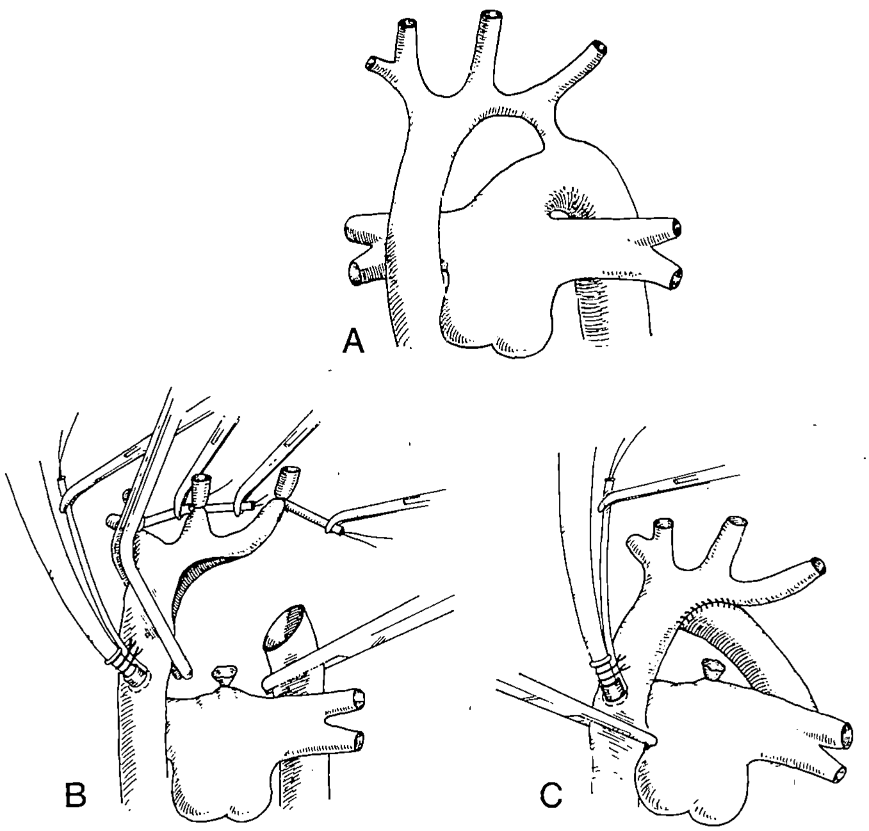

Fig 1. Operative technique for arch repair using isolated myocardial perfusion. (A) Preoperative anatomy of coarctation of the aorta with hypoplastic aortic arch. (B) Placement of ascending aortic clamp distal to the aortic cannula to perfuse the myocardium during arch repair. Head vessels are snared. (C) Completed arch repair.

Table 1. Clinical Patient Data

Patient No.	Age at Operation	Weight at Operation (kg)	Diagnosis	Procedure	Myocardial Ischemic Time (min)	Cerebral Ischemic Time (min)			Result
						During Arch Reconstruction	During Intracardiac Repair	Total	
1	1 mo	3.9	CoA, hypoplastic aortic arch, DORV, subpulmonary VSD, ASD	Arch repair, arterial switch, VSD and ASD closure	86	16	38	54	Well
2	3 mo	3.4	Hypoplastic aortic arch, DORV, subpulmonary VSD, ASD, SAS, ASD, S/P coarctation repair	Arch repair, arterial switch, aortic valvotomy, resection of subaortic muscle, VSD and ASD closure	77	41	. . .	41	Well
3	2 days	3.3	IAA (B), SAS, VSD, Di George syndrome	Arch repair, resection of subaortic muscle, VSD closure	37	23	32	55	Well
4	17 days	3.2	CoA, hypoplastic aortic arch, AS	Arch repair, aortic valvotomy	16	20	. . .	20	Well
5	6 days	3.2	CoA, hypoplastic aortic arch, DORV, subpulmonary VSD, SAS, ASD	Arch repair, arterial switch, VSD and ASD closure	71	16	29	45	Well

AS = aortic stenosis; ASD = atrial septal defect; CoA = coarctation of the aorta; DORV = double-outlet right ventricle; IAA = interrupted aortic arch; SAS = subaortic stenosis; VSD = ventricular septal defect.

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Comparative Study of Cell Saver and Ultrafiltration Nontransfusion in Cardiac Surgery

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Hemoconcentration for the establishment of no-donor blood transfusion in open heart surgery was assessed in regard to both the saving of protein and platelets and the exclusion of free hemoglobin. Two different types of hemoconcentrator were compared: the ultrafilter (group I, 6 patients) and the Cell Saver (group II, 6 patients). The total serum protein level, expressed as the percent recovery of the preoperative value, after hemoconcentration was significantly higher in group I (group I versus group II: total serum protein, 118% versus 87% [$p < 0.05$]; fibrinogen, 77% versus 50% [$p < 0.01$]; immunoglobulin, 83% versus 60% [$p < 0.01$]). The platelets also seemed to be well preserved after hemoconcentration in group I. Although the exclusion of free hemoglobin from plasma was inferior in group I compared with group II, the postoperative plasma free hemoglobin level did not increase in group I. We conclude that use of the Cell Saver in nontransfusion cardiopulmonary bypass might cause a severe depletion of various proteins and that the ultrafilter is both safer and more useful if employed routinely.

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The importance of blood conservation has been emphasized as a means to prevent occurrences of posttransfusion hepatitis as well as other blood-mediated infections. By improving existing methods [1–3], more than 60% of open heart procedures could be accomplished without transfusions of donor blood [4]. The development of blood concentration techniques seems to be a promising and effective means of performing open heart operations without transfusing blood.

Two types of blood concentrators, the ultrafilter and the Cell Saver, have recently become available. Both efficiently concentrate red blood cells, but otherwise, each has advantages and disadvantages. The ultrafilter provides a sufficient saving in protein. Theoretically, any blood component whose molecular weight is either almost the same as or greater than that of albumin can be preserved by this method [5]. However, it fails to exclude the plasma free hemoglobin [6], which is produced by hemolysis and toxic to the kidneys [7], because its molecular weight is almost identical to that of albumin. Consequently, transfusion of the ultrafiltrated blood can impair postoperative renal function.

The Cell Saver, on the other hand, is a centrifugal type of blood concentrator. It concentrates the blood by removing plasma from whole blood [8]. Thus, this method efficiently excludes free hemoglobin, but it causes a loss of serum protein. Usually, such protein depletion can be replaced by a blood transfusion.

The question facing cardiac surgeons is which of these two blood concentrators is preferable for nonblood-prime cardiopulmonary bypass (CPB). The purpose of this study was to assess and compare the efficacy of both types of hemoconcentrators during open heart operations without donor blood transfusions.

Material and Methods

Twelve patients underwent a nontransfusion open heart operation using a hemoconcentrator. The patients were randomly divided into two equal groups. For group I, an ultrafilter (Hemoconcentrator; C.R. Bard, Inc, Santa Ana, CA) was used and in group II, the Cell Saver III (Haemonetics Corporation, Braintree, MA). In all patients, CPB pump was primed with 500 mL of human protein solution, 5 g/100 mL, plus crystalloid solution. The crystalloid cardioplegic solution comprised the following: Na, 80 mEq/L; K, 20 mEq/L; Ca, 0.2 mEq/L; HCO_3, 10 mEq/L; glucose, 25 g/L; insulin, 5 U/L; and osmolarity, 340 mosm/L. It was administered every 20 minutes during aortic cross-clamping in an initial dose of 10 mL/kg and additional doses of 5 mL/kg. Ice slush was used for topical cooling. Those solutions were not discarded by wall suction, but were returned to the cardiotomy reservoir and were contained in the calculation of water balance in CPB.

Hemoconcentration by Ultrafilter

The ultrafilter was inserted in parallel with the bypass circuit between the venous reservoir and the cardiotomy reservoir (Fig 1). Ultrafiltration was performed at a rate of 300 mL/min of bypass flow and at 450 mm Hg of evacuation pressure during CPB to exclude the surplus crystalloid fluid of the cardioplegic or topical cooling solution.

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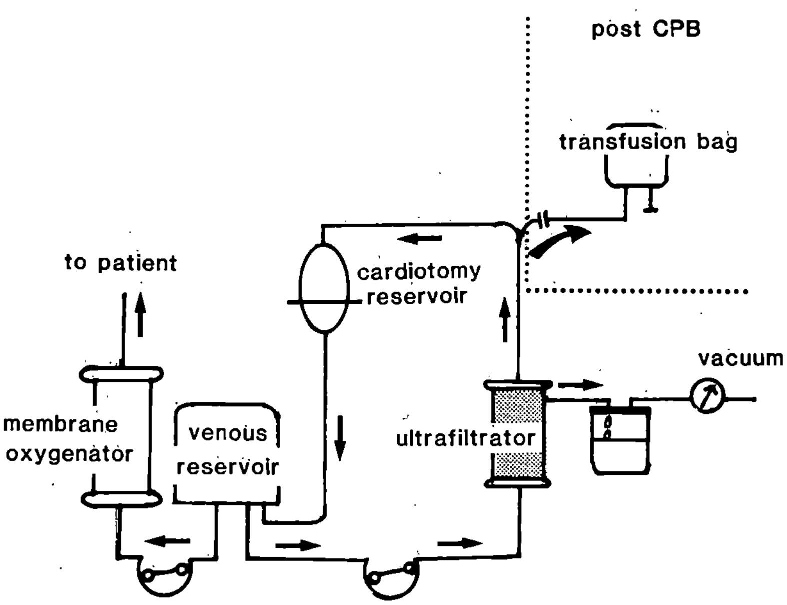

Fig 1. The ultrafilter is connected between the venous reservoir and the cardiotomy reservoir. The amount of filtration is controlled by the rate of flow through the roller pump and the evacuation pressure. After cardiopulmonary bypass (CPB), *the single-pass ultrafiltration method was effective for immediate transfusion.*

After the termination of CPB, a single-pass ultrafiltration method [9] was used at a rate of 150 mL/min of bypass flow and at 550 mm Hg of evacuation pressure for the hemoconcentration of the dilute blood remaining in the extracorporeal circuit. The ultrafiltrated blood was transferred to the bags and then immediately transfused.

Hemoconcentration by Cell Saver

The Cell Saver was used only after CPB because one centrifugation requires 500 mL of blood volume. After CPB, the entire contents of the cardiopulmonary circuit were drawn through a Y in the arterial line. The centrifugation speed was kept at 5,650 rpm. The supernatant (plasma and prime) was discarded during centrifugation into a waste bag. The concentrated blood was then transferred to the bags for intravenous infusion.

Data Acquisition

The values of the following variables were obtained before, during, at the end of CPB, and just after transfusion of the concentrated blood: hematocrit, total serum protein, platelets, and plasma free hemoglobin. Plasma fibrinogen and immunoglobulins were measured preoperatively and after transfusion of the concentrated blood.

Pulmonary function, postoperative blood loss, and renal function were also evaluated. Pulmonary function was assessed by perioperative changes (preoperative value, value after intubation in the operating room, postoperative value, value just after return to the intensive care unit) in the respiratory index (R index) [10]. The R index is calculated by the following equation:

$$[(P_B - PH_2O)\ FiO_2 - PaCO_2 - PaO_2] / PaO_2$$
$$= A\text{-}aDO_2 / PaO_2,$$

where P_B = barometric pressure (760 mm Hg), PH_2O = alveolar water vapor pressure (approximately 47 mm Hg), FiO_2 = fractional concentration of oxygen in inspired gas, $PaCO_2$ = arterial carbon dioxide tension, PaO_2 = arterial oxygen tension, and $A\text{-}aDO_2$ = alveolar-arterial oxygen pressure difference.

The $A\text{-}aDO_2$ is a representative indicator of the physiological shunt caused by pulmonary insufficiency. However, it would not be an adequate factor in most clinical environments because FiO_2 is variable. In contrast, the R index, in which $A\text{-}aDO_2$ is divided by PaO_2, is not affected by the change in FiO_2. This index is useful for the estimation not only of oxygenation efficacy of the lung but also of the prognosis of respiratory failure. An R index of 0.1 to 0.37 is considered normal; a patient with an R index of greater than 2.0 requires intubation. We usually use this index as one of the variables for weaning the patient from the respirator (R index < 1.0) after a cardiac operation.

Renal function was assessed according to perioperative changes (preoperative, postoperative day 1) in serum creatinine levels, blood urea nitrogen levels, and creatinine clearance.

Data Analysis

The levels of blood components such as platelets, total serum protein, fibrinogen, and immunoglobulins (IgG, IgA, IgM) after hemoconcentration were determined as percent recovery of preoperative values by the equation $100 \times (S2/S1)$, where S1 = the preoperative value, and S2 = the value just after transfusion of the concentrated blood, corrected by the hematocrit value to minimize the influence of hemodilution at this time (S2 = observed value × [postoperative hematocrit/preoperative hematocrit]). The efficacy of the hemoconcentrator to exclude free hemoglobin from the plasma was expressed as follows: $100 \times (F2 - F1)/F1$ where F1 = the value of serum free hemoglobin at the end of CPB, and F2 = the value of serum free hemoglobin after transfusion of the concentrated blood; F2 was also corrected by the hematocrit value.

Table 1. Summary of Patient and Operative Data[a,b]

Variable	Group I (Ultrafilter) (n = 6)	Group II (Cell Saver) (n = 5)
Age (yr)	42 ± 8	35 ± 20
Body weight (kg)	51 ± 6	52 ± 6
Procedure		
ASD closure	3	2
VSD closure	1	1
AVR	1	2
MVR	1	1
CPB time (min)	93 ± 49	120 ± 53
Fluid removed (mL)	1,608 ± 555	1,456 ± 465
Fluid balance (mL)	+1,274 ± 810	+1,626 ± 185

[a] Where applicable, data are shown as the mean ± the standard deviation. [b] There were no significant differences between the groups.

ASD = atrial septal defect; AVR = aortic valve replacement; CPB = cardiopulmonary bypass; MVR = mitral valve replacement; VSD = ventricular septal defect.

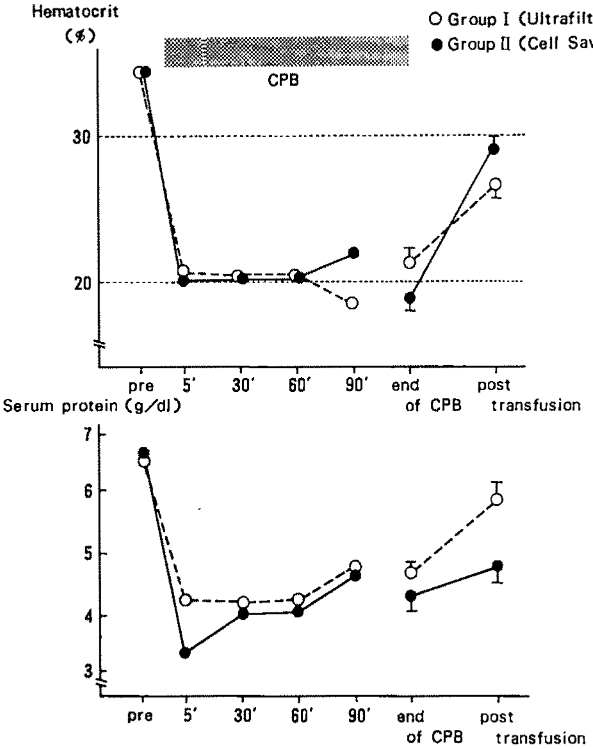

Fig 2. Changes in hematocrit and serum protein levels. In group I, restoration of serum protein after transfusion (28% increase) was parallel to that of hematocrit (27% increase), whereas in group II there was poor restoration of serum protein (16% increase) compared with hematocrit (56% increase). Data at the end of cardiopulmonary bypass (CPB) and after transfusion are shown as the mean ± the standard error of the mean.

Student's t tests for paired and unpaired data differences were used for statistical analysis. The results are presented as the mean ± the standard deviation unless stated otherwise.

Results

The group profiles are shown in Table 1. There were no significant differences between the two groups with respect to age, body weight, CPB time, fluid removed, and fluid balance.

Recovery of Blood Components

During CPB, the hematocrit decreased to 20% in both groups (Fig 2). After transfusion of the concentrated blood, the hematocrit increased to 27% in group I and 29% in group II. The total serum protein level at the end of CPB was 4.6 g/dL in group I and 4.2 g/dL in group II. After transfusion, it increased to 5.9 g/dL in group I and 4.85 g/dL in group II (see Fig 2).

The percent recovery of total serum protein was approximately 100% in group I (118.4% ± 8.0%) because of the human protein solution in the pump prime (Table 2). In contrast, the percent recovery in group II was 86.7% ± 11.5% ($p < 0.05$ versus group I). The percent recovery of platelets was 69.0% ± 24.7% in group I and 48.0% ± 13.3% in group II ($p = 0.1$) (see Table 2). The percent recovery of fibrinogen was significantly better in group I (76.7% ± 11.8% versus 50.0% ± 13.6% [n = 5]; $p < 0.01$) (see Table 2). The percent recovery of immunoglobulins (IgG, IgA, IgM) was also significantly better in group I: IgG, 82.8% ± 14.1% versus 60.0% ± 6.8% ($p < 0.01$): IgA, 85.0% ± 17.2% versus 58.2% ± 4.8% ($p < 0.01$); and IgM, 84.7% ± 16.0% versus 60.1% ± 13.1% ($p < 0.05$) (see Table 2).

Exclusion of Plasma Free Hemoglobin

The free hemoglobin level increased linearly in both groups during CPB, and at the end of CPB it reached 47.1 ± 42.8 mg/dL in group I and 70.9 ± 35.5 mg/dL in group II (Fig 3). The exclusion of free hemoglobin was accomplished efficiently by the Cell Saver; 36.3% ± 11.1% of free hemoglobin contained in the postoperative pump blood was excluded in group II versus 6.65% ± 27.1% in group

Table 2. Preservation of Blood Components and Exclusion of Plasma Free Hemoglobin[a]

Variable	Group I	Group II	p Value
Recovery of blood components (% recovery[b])			
Total serum protein	118.4 ± 8.0	86.7 ± 11.5	<0.05
Platelet	69.0 ± 24.7	48.0 ± 13.3	≒0.1
Fibrinogen	76.7 ± 11.8	50.0 ± 13.6	<0.01
Immunoglobulin			
IgG	82.8 ± 14.1	60.0 ± 6.8	<0.01
IgA	85.0 ± 17.2	58.2 ± 4.8	<0.01
IgM	84.7 ± 16.0	60.1 ± 13.1	<0.05
Exclusion of free hemoglobin (% change[c])	−6.65 ± 27.1	−36.3 ± 11.1	<0.05

[a] Data are shown as the mean ± the standard deviation. [b] This is the percent recovery from the prebypass level. [c] This is the percent change in the serum level after transfusion of hemoconcentrated blood.

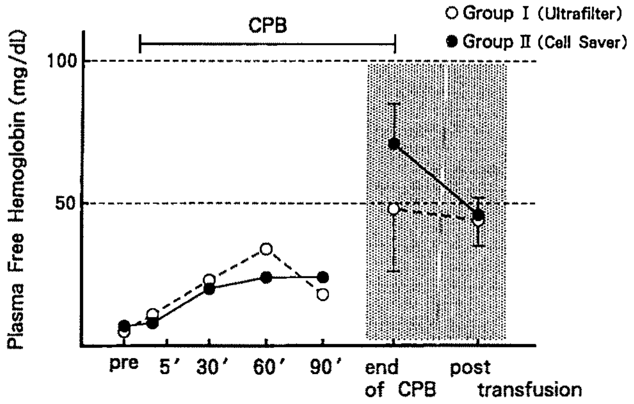

Fig 3. Major changes in plasma free hemoglobin. In both groups, free hemoglobin increased linearly during cardiopulmonary bypass (CPB). *At the end of CPB, it was relatively high in group II because of the length of bypass. However, the Cell Saver effectively reduced the level. Data at the end of CPB and after transfusion are shown as the mean ± the standard error of the mean.*

I ($p < 0.05$) (see Table 2). The serum free hemoglobin level of both groups did not alter after the concentration and transfusion of the blood remaining in the extracorporeal circuit (46.6% ± 43.9 mg/dL versus 44.3 ± 23.6 mg/dL, group I versus group II).

Postoperative Blood Loss and Pulmonary and Renal Function

Postoperative blood loss from chest drainage was 11.2 ± 3.4 mL/kg in group I and 12.0 ± 8.3 mL/kg in group II (Table 3). There were no significant differences between the two groups. The R index did not show any significant perioperative changes (group I: preoperative, 0.43 ± 0.37, and postoperative, 0.36 ± 0.14; group II: preoperative, 0.32 ± 0.36, and postoperative, 0.21 ± 0.22). The serum blood urea nitrogen level, serum creatinine level, and creatinine clearance on the first postoperative day showed no significant perioperative changes in either group (see Table 3).

Comment

Various kinds of viral infections, such as posttransfusion hepatitis, adult T-cell leukemia, and acquired immunodeficiency syndrome, can be transmitted by blood transfusions [11, 12]. Graft versus host disease after blood transfusion [13] has become a problem in cardiac surgery. At the Cleveland Clinic, many efforts have been made over the past few years to reduce the amount of blood required, and as a result, at present, approximately 60% of all their cardiac patients receive no blood at all during hospitalization [4]. Hemoconcentration in open heart operations done without blood transfusions has some benefits because the transfusion of all the priming solution sometimes gives rise to an excessive volume overload, resulting in heart failure and tissue edema [14–16]. As a result, hemoconcentration during CPB has become an essential method in nonblood-prime CPB, as critical hemodilution (a hematocrit lower than 15% [17]) can occur because of the addition of crystalloid solutions, such as cardioplegia, and topical solutions [18]. Especially in patients with renal failure, the use of a hemoconcentrator during bypass enables us to control the balance between water and electrolytes [19].

Two types of hemoconcentrators, the ultrafilter and the Cell Saver, are available for open heart operations. The ultrafilter was introduced in CPB in 1984 by Magilligan and Oyama [20]. Across its semipermeable membrane, water, electrolytes, and small molecules are transported by hydrostatic pressure. Large molecular substances such as blood cells and large parts of protein including albumin, fibrinogen, and immunoglobulins remain well preserved. To minimize the risk of postoperative bleeding and infection, it is important to save these blood cells and protein. The loss of albumin reduces colloid osmotic pressure and can induce postoperative pulmonary dysfunction [14]. The loss of platelets and fibrinogen can result in postoperative bleeding. The loss of immunoglobulins can affect the postoperative host-defense mechanism.

In contrast to these major conservation effects, the usual ultrafilter fails to remove the free hemoglobin [6] because its molecular weight is almost identical to that of albumin. After nontransfusion CPB, all of the blood remaining in the CPB circuit must be returned to the patient. If a free hemoglobin–enriched solution is transfused in this way, it is not possible to reduce the negative influence on postoperative renal function.

The Cell Saver is beneficial for the exclusion of hemolytic products including free hemoglobin or other particles. Blood aspirated from the surgical field can be safely returned with this apparatus [21]. However, as both blood components in addition to red blood cells are disposed of, excessive use of the Cell Saver can cause a depletion of either platelets or serum protein. In operations without transfusion of blood, blood components lost are not supplied, and this can lead to the postoperative compli-

Table 3. Comparison of Preoperative and Postoperative Data[a,b]

	Group I		Group II	
Variable	Preop	Postop	Preop	Postop
Respiratory index	0.43 ± 0.37	0.36 ± 0.14	0.32 ± 0.36	0.21 ± 0.22
BUN (mg/dL)	12.8 ± 3.8	14.0 ± 3.8	16.8 ± 3.3	20.5 ± 9.60
Creatinine (mg/dL)	0.82 ± 0.21	0.92 ± 0.24	1.02 ± 0.30	1.08 ± 0.44
Creatinine clearance (mL/min/BSA)	71.0 ± 16.0	61.6 ± 5.0	65.8 ± 7.7	60.9 ± 22.0
Blood loss (mL/kg)	. . .	11.2 ± 3.42	. . .	12.0 ± 8.35

[a] Data are shown as the mean ± the standard deviation. [b] There were no significant differences between the groups preoperatively or postoperatively.

BSA = body surface area; BUN = blood urea nitrogen.

cations already mentioned. In this study, we tested both the advantages and disadvantages of the two blood concentrators in nontransfusion CPB.

The present study clearly showed the significant depletion of blood components by the Cell Saver during nontransfusion cardiac operations. Platelets decreased to 48% of the prebypass value in group II (69% in group I), fibrinogen decreased to 50% in group II (77% in group I), and immunoglobulins also decreased to about 60% of the prebypass value. In no patient in this study did pulmonary dysfunction, bleeding tendency, or major infection develop postoperatively, but the significant depletion of the functional proteins discourages us from recommending use of the Cell Saver in open heart procedures performed without transfusing blood.

On the other hand, the Cell Saver removed the plasma free hemoglobin efficiently. The exclusion of free hemoglobin at hemoconcentration was 6.65% in group I and 36.3% in group II. These data, however, seem to suggest a major disadvantage to use of the ultrafilter in nontransfusion CPB because the plasma free hemoglobin level after transfusion of the concentrated blood did not exceed the pretransfusion level (12% decrease in pretransfusion value). As Savitsky and associates [7] demonstrated, depression of renal function by free hemoglobin is reversible as long as there is no persistent hemolysis and no preexisting renal dysfunction. Indeed, there was no consecutive hemolysis in group I and no significance between the preoperative and postoperative renal function in this group. In general, free hemoglobin combines immediately with plasma haptoglobulin [22] and is metabolized in the liver. If the production of free hemoglobin exceeds this capacity, it is filtrated through the kidney, which results in renal dysfunction. Therefore, ultrafiltration can be safely performed if there is no remarkable hematuria in nontransfusion CPB.

Taking the various advantages and disadvantages of the hemoconcentrators into account, we now routinely use both the ultrafilter and the Cell Saver for a number of patients during open heart operations to avoid transfusion of donor blood. To date, no postoperative complications related to their use have been observed. We perform ultrafiltration during and after bypass for the hemoconcentration of dilute pump blood; employment of the Cell Saver has been limited to the salvaging of blood shed through discard suction or to the cleaning of severely damaged blood. Thus, the best method of hemoconcentration may be a combination of both.

Cost savings should also be considered when selecting hemoconcentrators. In Japan, the one-time cost of ultrafiltration with the ultrafilter is $286, which is less than that for the Cell Saver, whose disposable cell-washing circuit costs $329 (calculated at $1 = 140 yen). Moreover, ultrafiltration can be performed with an evacuation pump only ($460), whereas the Cell Saver (Cell Saver III) is vastly more expensive ($64,000).

Not only the hemoconcentration device, but also the difference in protein-saving efficacy should be evaluated in cost savings. The present study suggests that use of the Cell Saver results in a protein loss approximately 25% inferior compared with ultrafiltration. The serum protein loss reaches 600 mL in a 50-kg patient (hematocrit, 40%). If it is replaced by 3 packs of fresh frozen plasma (1 pack = $31), the protein saving by ultrafiltration means a cost savings of $93. Therefore, we believe the ultrafiltration method is preferable, and the presence of minor hemolysis should not outweigh its advantages as an important protein-saving device.

We draw two major conclusions from this study.

1. Use of the Cell Saver as a hemoconcentrator in CPB without donor blood induces a severe deficiency in both serum protein levels and platelet counts.
2. The ultrafilter is preferable for hemoconcentration in open heart operations performed without transfusion of donor blood when the patient has neither severe hemolysis nor preoperative renal dysfunction.

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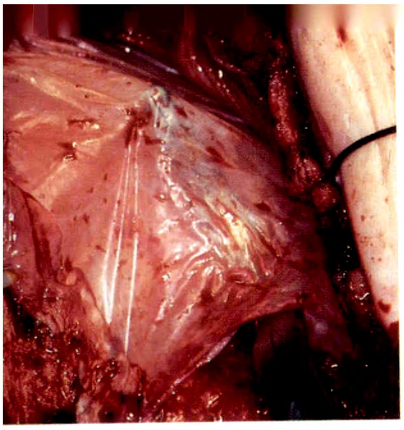

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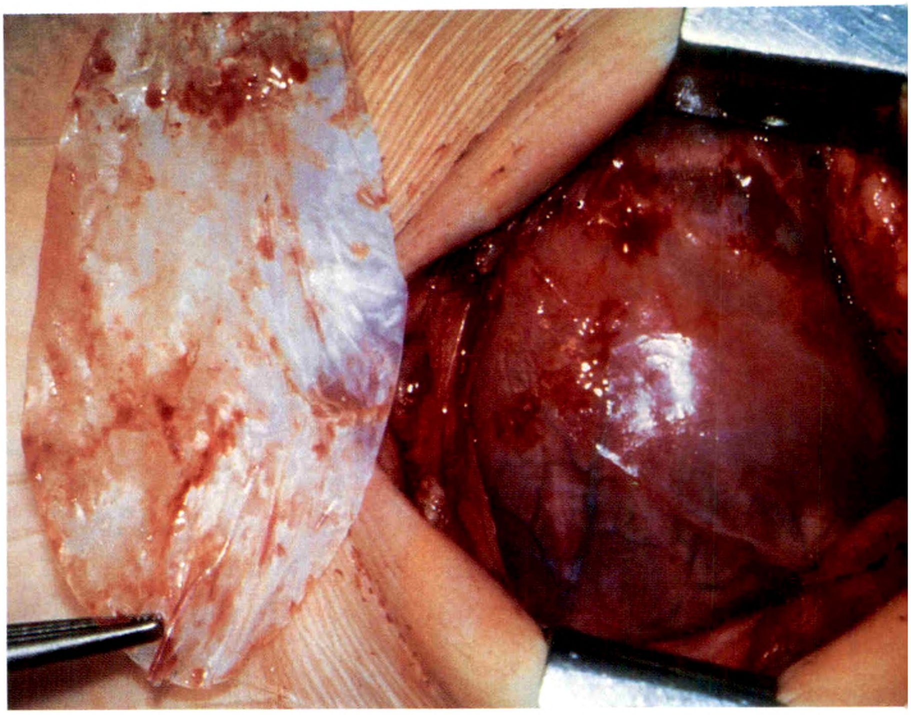

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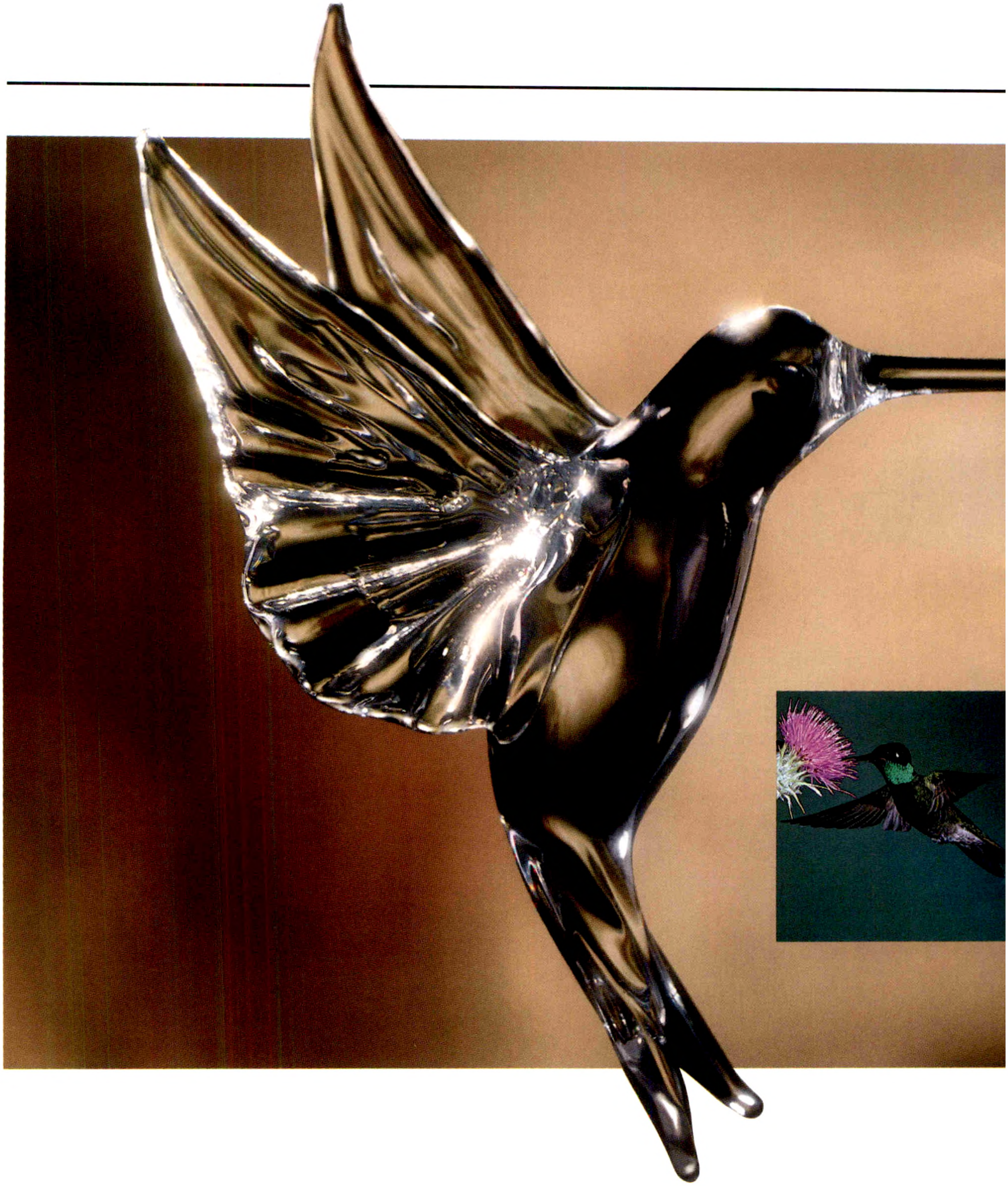

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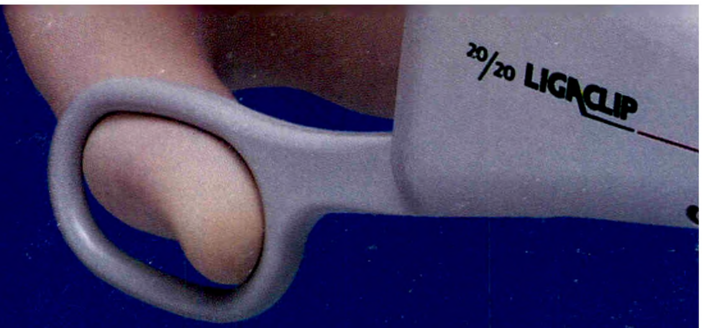

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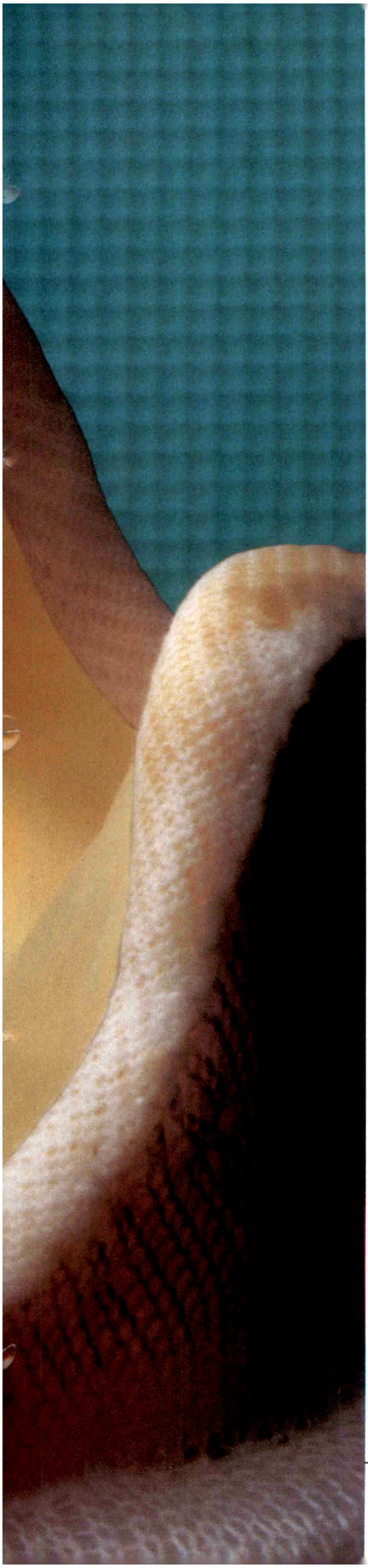

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The decision to use a tissue valve must ultimately be made by the physician on an individual basis after a careful evaluation of the short- and long-term risks and benefits to the patient are made, and alternative methods of treatment are considered. Overall durability, especially long-term, has not been established for bioprostheses. Serious complications, sometimes leading to replacement of the valve and/or death, may be associated with the use of prosthetic valves (see Complications). A full explanation of the benefits and risks should be given to each prospective patient before surgery. **Note:** Bioprostheses should be used with caution in the presence of severe systemic hypertension or when the anticipated patient longevity is longer than the known longevity of the prosthesis. Careful and continuous medical follow-up (at least by an annual visit to the physician) is advised so that valve-related complications, particularly those related to material failure, can be diagnosed and properly managed.Recipients of prosthetic heart valves who are undergoing dental procedures should receive prophylactic antibiotic therapy to minimize the possibility of prosthetic infection. Bioprosthetic heart valve recipients should be maintained on anticoagulant therapy, except where contraindicated, during the initial healing stages after implantation, approximately 2 to 3 months. Anticoagulants should then be discontinued over a period of 10 days, except in those patients for whom indefinite anticoagulant protection is indicated, i.e., in the absence of sinus rhythm and in patients with a dilated left atrium, calcification of the atrial wall or history of previous atrial thrombosis. The appropriate anticoagulation therapy must be determined by the physician on an individual basis. **Adequate rinsing with physiological saline, as described in the Technique section, is mandatory before implantation to reduce the glutaraldehyde concentration. No other solutions, drugs, chemicals, antibiotics, etc. should ever be added to the glutaraldehyde or rinse solutions, as irreparable damage to the leaflet tissue, which may not be apparent under visual inspection, may result. The Carpentier-Edwards bioprostheses must be kept moist at all times. Drying out will cause irreversible damage to the leaflet tissue. To prevent drying out during implantation, the valves should be irrigated periodically on both sides with sterile physiological saline.** Passing diagnostic catheters and transvenous pacing leads across a Carpentier-Edwards bioprosthesis could damage the valve. Consequently, it is recommended that these procedures be avoided. Both the valve and glutaraldehyde storage solution are **sterile.** The outside of the jar is **not sterile** and must **not** be placed in the sterile field. Furthermore, the valve and container should **never** be subjected to sterilization procedures involving ethylene oxide, propylene oxide, steam, or irradiation. Storage between 10°C and 25°C is recommended; however, care should be exercised to avoid freezing or extreme heat, which may damage the valvular tissue. **Any valve that has been frozen or exposed to temperatures greater than 60°C should not be used for human implantation.** Glutaraldehyde may cause irritation of the skin, eyes, nose, and throat, and may also cause skin sensitization. Avoid prolonged or repeated contact or prolonged breathing of the vapor. Use only with adequate ventilation. In the event of contact, immediately flush the affected area with water. In the event of contact with eyes, seek medical attention. **Surgical Precautions: 1.** When choosing a valve for a given patient, the size, age, and physical condition of the patient in relation to the size of the prosthesis must be taken into consideration to minimize the possibility of obtaining a suboptimal hemodynamic result. The selection of a valve, however, must ultimately be made by the physician on an individual basis after carefully weighing all of the risks and benefits to the patient. **2.** Due to the relative flexibility of the frame, care must be exercised to avoid the use of too large a prosthesis to prevent folding or extreme deformation of the stent that may render the valve incompetent. Oversizing may create highly localized mechanical stresses resulting in tissue failure in the form of detachment of the leaflets or porcine aortic wall from the valve stent posts. **3.** Left ventricular perforation or ulceration has been reported. Both open and closed chest cardiac massage in patients with an open strut mitral prosthesis increases the risk of ventricular perforation. Additionally, lifting, flexing, and manual compression of the distended heart after discontinuation of bypass may cause a commissure strut to be pushed through the endocardium, producing a dissecting hematoma with subsequent uncontrolled hemorrhage. **4.** The spacing of the sutures in the remnant of the valvular orifice and the prosthesis suture ring must be carefully matched to avoid folding of the leaflets or extreme distortion of the orifice. Edwards CVS Division has received reports in which individual mattress sutures, spanning a distance of 10 to 15mm, produced a purse-string effect causing compression of the valve orifice. **5.** When using interrupted sutures, it is important to cut the sutures close to the knots and to ensure that exposed suture tails will not come into contact with the leaflet tissue. Cases have been reported in which bioprostheses developed severe regurgitation and had to be replaced as a result of wear due to contact with sutures. **6.** Unlike rigid mechanical valves, the stent wall is soft and will not resist needle penetration. Accordingly, extreme care must be exercised when placing sutures through the sewing margin to avoid penetration of the side wall of the stent and possible laceration of the leaflet tissue. **7.** As with all prostheses that have open cages, free struts, or commissure supports, care must be exercised, particularly in mitral replacement, to avoid looping or catching a suture around the free legs which would interfere with proper valvular function. **8.** The stent of the bioprosthesis is asymmetrical to conform to the natural shape of the porcine aortic valve. Consequently, the commissure supports are not equidistantly spaced. The contrasting suture markers in the sewing ring of the mitral valve denote the greatest intercommissural distance. These two commissure supports should be oriented so as to straddle the ventricular outflow tract to avoid murmurs due to blood flow turbulence. **Note:** The contrasting suture material should not be removed from the prosthesis. **9.** In the aortic position, the struts should correspond to the remnants of the natural commissures so as not to obstruct the coronary ostia. **10.** A serial number tag is attached to the sewing ring of each valve by a suture. This serial number should be checked against the number on the jar; if any difference is noted, the valve should be returned unused. This tag should not be detached from the valve until implant is imminent. Care should be exercised to avoid cutting or tearing the suture ring cloth during removal. **11.** Gentle handling is required for all implantable devices. If the valve is dropped, damaged, or mishandled in any way, it must not be used for human implantation. **Complications:** As with all prosthetic heart valves, serious complications, sometimes leading to death, may be associated with the use of tissue valves. In addition, complications due to individual patient reaction to an implanted device, or to physical or chemical changes in the components, particularly those of biological origin, may occur at varying intervals (hours or days) necessitating reoperation and replacement of the prosthetic device. Complications associated with the use of Carpentier-Edwards bioprostheses include stenosis, regurgitation through an incompetent valve, perivalvular leak, endocarditis, hemolysis, thromboembolism, thrombotic obstruction, bleeding diatheses related to the use of anticoagulant therapy, ventricular perforation by stent posts, looped sutures, and malfunctions of the valve due to distortion at implant or physical or chemical deterioration of valve components. Types of tissue deterioration include infection, calcification, thickening, perforation, suture abrasion, instrument trauma, and leaflet or aortic wall detachment from the valve stent posts. These complications may present clinically as abnormal heart murmur, shortness of breath, exercise intolerance, dyspnea, orthopnea, anemia, fever, arrythmia, hemorrhage, transient ischemic attack, stroke, paralysis, low cardiac output, pulmonary edema, congestive heart failure, cardiac failure, and myocardial infarct. **Note:** Based on reports in the literature on tissue valves, there appears to be an increased incidence of leaflet calcification in patients under the age of 20. In this regard, animal research studies show that a high systemic calcium level can lead to early calcification. Furthermore, at least one published report describes an apparent causal relationship between the consumption of daily calcium supplements and early leaflet calcification in an adult. When feasible, repeated intravenous injections containing calcium should be avoided during the postoperative period; and excessive milk or dairy product consumption should be avoided in children. There are no clinical data presently available demonstrating increased resistance of Carpentier-Edwards bioprostheses to calcification as compared to other commercially available porcine valves. **Caution:** Federal (USA) law restricts this device to sale by or on the order of a physician. Baxter International Inc. is the owner of the following US patent which covers the products described herein: 4,106,129. Patent numbers in foreign countries supplied upon request. See package insert accompanying product for a full discussion of warnings, contraindications, complications and prescribing information. Carpentier-Edwards® is a registered trademark of Baxter International Inc.

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Life-Threatening Acute Posterior Mediastinitis Due to Esophageal Perforation

Clay M. Burnett, MD, Alexander S. Rosemurgy, MD, and Eric A. Pfeiffer, BS

Department of Surgery, University of South Florida, Tampa, Florida

We analyzed the treatment of a recent group of patients with life-threatening acute posterior mediastinitis due to esophageal perforation to elucidate common factors in successful treatment. Life-threatening acute posterior mediastinitis due to esophageal perforation was diagnosed in 16 patients over the past 12 years. Esophageal perforation resulted from endoscopy in 11 patients, retching in 4, and blunt trauma in 1 patient. Preoperative serum albumin levels were higher in patients who survived. Fourteen of 16 patients (88%) underwent exploration: mediastinal drainage in 14 (10 survived), esophageal repair in 9 (7 survived) with diversion in 3 (3 survived), and stent placement in 2 (1 survived). Six of 16 patients (38%) died, always of polymicrobial sepsis. Female patients and those with cancer, endoscopic perforations, delayed diagnosis, persistent mediastinal contamination, mediastinal suppuration or necrotizing cellulitis, and postoperative complications did poorly. Antibiotics must be effective against both gram-positive and gram-negative bacteria, and against both anaerobic and aerobic bacteria. Early surgical intervention is key, particularly elimination of ongoing mediastinal soilage. Thorough mediastinal debridement and wide mediastinal drainage appear to be important in improving survival of patients with life-threatening acute posterior mediastinitis due to esophageal perforation.

(Ann Thorac Surg 1990;49:979–83)

Interest in nonsurgical management of esophageal perforation is growing. This approach appears quite successful in patients with uncomplicated perforations, those associated with minimal contamination and a paucity of signs and symptoms [1]. Esophageal perforation accompanied by mediastinal contamination, however, is generally believed to require surgical management to avoid or control posterior mediastinitis [2]. This management may consist of mediastinal drainage, esophageal repair or resection, esophageal diversion, antimicrobial therapy, and nutritional supplementation.

Previous studies of posterior mediastinitis have compared patients with many different underlying diseases, with varying severity and origin of contamination, with varying degrees of mediastinitis, and with wide variations in general health and clinical condition. Unfortunately, such a wide range of poorly controlled variables makes any comparative analysis of treatment factors difficult. This retrospective study was undertaken to address some of these criticisms by analyzing treatment and outcome in a well-defined population of critically ill patients with severe, life-threatening acute posterior mediastinitis due exclusively to esophageal perforation.

Material and Methods

Hospital, clinic, and physician records were used to compile data from patients of the Department of Surgery, University of South Florida, who over the past 12 years had posterior mediastinitis. Medical records were reviewed to accumulate and analyze demographic data, presenting symptoms, cause of esophageal perforation, underlying disease states, diagnostic modalities undertaken, endoscopic findings, laboratory abnormalities, operative findings, surgical management, bacteriological testing, complications, and outcome. Patients with esophageal perforations were considered appropriate for this study only if they had documented posterior mediastinitis and their general condition, clinical findings, and degree of mediastinitis were consistent with a "critically ill" designation; all patients in this study were symptomatic, required intensive care unit invasive monitoring, had evidence of sepsis with multisystem organ failure, and had radiological or operative evidence of mediastinitis. Therefore, asymptomatic patients, patients not ill, and patients with minor perforations diagnosed by limited extravasation of radiological contrast were excluded.

Results

Acute life-threatening posterior mediastinitis due to esophageal perforation occurred in 16 patients, 12 male and four female, over the past 12 years. Esophageal perforations and posterior mediastinitis were documented in all patients. The average age of the 16 patients was 55.3 ± 3.6 years (standard error of the mean). The average age of the male patients was 53.3 ± 4.2 years, and that of the female patients was 61.2 ± 5.3 years.

All patients were symptomatic. Chest pain, shortness of breath, nausea, and malaise were frequently reported, though not universally. Fifteen of the 16 patients underwent radiological esophageal contrast examinations for diagnosis. The single patient operated on without such a

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contrast study was a 69-year-old man with Boerhaave's syndrome. His history, physical examination, and chest roentgenogram were believed to provide sufficient evidence to warrant prompt surgical exploration. In all 15 patients who underwent radiological esophageal contrast studies, esophageal perforations were documented. In the recent years of this review, computed tomographic scanning was used in 3 patients; each computed tomographic scan confirmed mediastinal suppuration.

Time between esophageal perforation and diagnosis for the 14 patients who underwent operation averaged 61.3 hours. For patients who survived hospitalization, the time between perforation and diagnosis averaged 29.1 ± 7.3 hours ($n = 10$). For only 2 of 10 survivors did more than 24 hours elapse between perforation and diagnosis. For patients who did not survive hospitalization after operation, the time between perforation and diagnosis averaged 115 ± 61 hours ($n = 6$).

Eleven (69%) of the 16 esophageal perforations resulted from endoscopy: 6 patients had benign strictures (2 died) and 5 patients had obstructing cancers (3 died). All endoscopies were performed by experienced gastroenterologists using flexible fiberoptic endoscopes. Eight of these 11 patients underwent concomitant esophageal dilation (5 died); esophageal perforation occurred in 3 patients during simple diagnostic endoscopy without dilation.

The cause of the esophageal perforations in the remaining 5 patients with severe acute posterior mediastinitis was postemetic esophageal perforation in 4 patients (1 died) and traumatic thoracic esophageal perforation due to blunt chest trauma in 1 patient.

The average white blood cell count at presentation for all 16 patients was $12.6 \pm 1.6 \times 10^9$/L. The white blood cell count at presentation in patients who survived hospitalization and those who did not survive hospitalization was $11.7 \pm 2.1 \times 10^9$/L and $14.3 \pm 2.4 \times 10^9$/L, respectively. Amylase levels at presentation, available in 7 patients, varied considerably among survivors and nonsurvivors: 71.4 ± 15.3 U/L ($n = 5$) versus 249.5 ± 233.3 U/L ($n = 2$). Serum albumin levels at presentation, available in 14 patients, averaged 3.20 ± 0.19 mg/dL. In patients who survived hospitalization, the average serum albumin level at presentation was 3.40 ± 0.23 mg/dL ($n = 8$), and in patients who did not survive hospitalization the average serum albumin level was 2.85 ± 0.29 mg/dL ($n = 6$) ($p < 0.05$; Student's t test).

Fourteen patients (88%) underwent therapeutic exploration. Methods of surgical management included mediastinal drainage ($n = 14$), closure of the esophageal perforation ($n = 9$), gastrostomy tube placement ($n = 8$), esophageal diversion ($n = 3$), esophageal stent placement ($n = 2$), and distal esophageal occlusion ($n = 1$). Of the 14 patients who underwent mediastinal drainage, 10 (71%) survived. Seven (78%) of 9 patients who underwent primary repair of their esophageal perforation survived. All 3 patients who underwent esophageal diversion survived: 1 underwent concomitant primary repair of the perforation and 1 underwent concomitant esophageal occlusion proximal to the perforation. One of 2 patients who underwent esophageal stent placement survived; the surviving patient underwent concomitant primary repair of his esophageal perforation, which was just distal to an obstructing esophageal cancer. Nine (82%) of 11 patients who underwent primary repair of or diversion away from their esophageal perforation survived. One of 3 patients who underwent operation without esophageal diversion or primary repair of the perforation survived. Two patients (12%), both gravely ill at the time of diagnosis of esophageal perforation and posterior mediastinitis and thus poor candidates for aggressive intervention, refused operation and died.

A host of other major surgical procedures were performed on the 14 patients undergoing therapeutic exploration. A feeding jejunostomy was performed in 9 patients (7 survived), tracheostomy was performed in 2 patients (1 survived), vagotomy and antrectomy with gastrojejunostomy were performed in 1 patient to treat a perforated gastric ulcer (the patient died), and vagotomy and pyloroplasty were performed in 1 patient to treat obstructing peptic ulcer disease (the patient survived).

Nine (64%) of the 14 patients who underwent operation had complications. All 5 patients who did not experience postoperative complications survived, whereas only 5 (56%) of the 9 patients who did experience postoperative complications survived. Complications included sepsis (4; 2 died), respiratory failure (3; 1 died), persistent extravasation from the esophageal perforation (2), renal failure (2; 2 died), perforated gastric ulcer (1; 1 died), delirium tremens (1; 1 died), cardiac failure (1; 1 died), serious cardiac arrhythmia (1), fatal aortoesophageal fistula (1), and esophagocutaneous fistula (1; 1 died).

In all, 6 (38%) of the 16 patients died after suffering esophageal perforation and posterior mediastinitis. Three of the 12 male and 3 of the 4 female patients died. The average age of the survivors and nonsurvivors was 54.5 ± 4.0 and 56.7 ± 6.6 years, respectively. The cause of death in each of the 6 patients was sepsis, although none of the 6 patients had evidence of an undrained mediastinal suppurative collection. Sepsis led to fatal aortoesophageal fistula in 1 patient and fatal multiorgan failure in 5 patients, 2 of whom were the patients who did not undergo operation.

All 14 patients who underwent surgical exploration had specimens of mediastinal fluid sampled at operation and processed for culture and sensitivity testing. Gram-positive and gram-negative bacteria and anaerobic and aerobic bacteria were cultured as well as yeast. The most common organisms cultured were *Streptococcus*, *Pseudomonas*, and *Candida* (Table 1). Three patients had cultures of mediastinal fluid sampled at operation that grew no organisms, 6 patients had cultures that grew from one to three organisms, and 5 patients had cultures that grew more than three organisms (Table 2). Two of 3 patients with negative cultures survived, 4 of 6 patients with from one to three organisms on culture survived, and 4 of 5 patients with more than three organisms on culture survived. The number of organisms grown in culture from samples obtained at operation was analyzed in light of the time that elapsed between esophageal perforation and diagnosis. For the 3 patients whose sample cultures produced no organisms less than 24 hours had elapsed

Table 1. Results of Cultures of Samples Obtained at Exploration

Organism	No. of Patients
Streptococcus	8
Pseudomonas	5
Candida	5
Escherichia coli	3
Enterobacter	3
Enterococcus	3
Staphylococcus	3
Bacteroides	2
Acinetobacter	1
Citrobacter	1
Klebsiella	1
Morganella	1
Proteus	1
Peptostreptococcus	1
Serratia	1
Veillonella	1
No growth	3

between the time of esophageal perforation and diagnosis. For 3 of 5 patients whose sample cultures yielded more than three organisms more than 48 hours had elapsed between the time of perforation and diagnosis (Table 2).

All patients were aggressively treated by multiple broad-spectrum antibiotic regimens. All required extended stay in the intensive care unit. The average length of hospitalization for the 6 patients who died was 11.8 days (range, four to 20 days), whereas for the 10 patients who survived the average was 40.8 days (range, 13 to 91 days).

Comment

Acute posterior mediastinitis is an uncommon disease with a substantial mortality, despite advances in surgery and critical care medicine [3]. Unfortunately, previous studies of acute posterior mediastinitis have compared patients of different time periods, of broad ranges in age, with many different underlying diseases, with varying causes of and degrees of mediastinitis, and with wide variations in clinical condition. This study addresses some of these criticisms by analyzing treatment and outcome using only a modern homogeneous population of patients with life-threatening acute posterior mediastinitis due only to confirmed esophageal perforation. The lengths of hospitalizations, uniformly involving intensive care unit stay, underscore the magnitude of illness faced by these patients.

Table 2. Number of Species Grown in Culture From Samples Obtained at Exploration Compared With Time Elapsed Between Perforation and Diagnosis

	No. of Organisms		
Time Elapsed (h)	0	1–3	>3
24	3	3	2
24–48	0	1	0
>48	0	2	3

Previous studies [4, 5] indicated that younger patients, particularly male patients, appear to enjoy a more favorable outcome after having esophageal perforation and posterior mediastinitis. Our data show no age difference between survivors and nonsurvivors. The male population of this review was generally younger, possibly helping to account for their lower mortality rate (25% versus 75%).

Endoscopic perforation is the major cause of esophageal perforation today. Most investigators believe that dilation therapy carries a greater risk of esophageal perforation than does endoscopy alone [6, 7]. This review documents a higher mortality rate for patients with esophageal perforation and mediastinitis caused by dilation relative to that caused by endoscopy alone: all endoscopic deaths in this report occurred in patients undergoing concomitant esophageal dilation.

Particularly ominous in this review was the presence of an underlying malignancy: 3 of 5 patients with underlying esophageal cancer died whereas only 3 of 11 patients without an underlying esophageal cancer died. Three of 4 women had an underlying esophageal cancer, again helping to explain the high mortality rate of women relative to men.

The results of laboratory testing were not unexpected. Serum albumin levels were generally depressed, reflecting the depleted visceral protein stores of this generally older population, of which 31% had cancer and an additional 31% had a clinically significant history of alcohol ingestion. Serum albumin levels were significantly higher in patients who survived. In accord with other reports [2, 8], this report shows the importance of nutritional health.

This study reports the belief that delay in diagnosis and definitive treatment continues to be a major factor influencing outcome after esophageal perforation [2, 3, 8]. In this study, early diagnosis and intervention limited mediastinal contamination and progression of mediastinitis and its complications, including death. If treatment is initiated within 24 hours after perforation, a successful outcome can be expected in patients in good general health; treatment delayed beyond 24 hours carries a progressively poorer prognosis. A prompt diagnosis is difficult, in part, because patient complaints are vague. A high degree of suspicion is necessary to diagnose esophageal perforation early; a detailed history is critical.

Esophagograms are generally recognized to be the safest and most accurate widely available diagnostic method [4, 9, 10] for the detection of esophageal perforation. Nonetheless, a 25% failure rate in localization by esophagogram has been reported [5, 9]. In this review, all patients had demonstration of their esophageal perforations by contrast esophagography when so tested, irrespective of the time from occurrence of perforation to contrast examination. Contrast esophagography initially should be performed with water-soluble contrast to detect gross defects, although we agree with other investigators that barium is the contrast agent of choice for detecting subtle injuries as it provides the best definition of esophageal anatomy [10]. Neither computed tomography nor magnetic resonance imaging has replaced esophagography in the evaluation of patients with possible esophageal perforation: esophagography detects more subtle perforations and anatomically localizes these perforations. Mediastinal imaging techniques help define the extent of mediastinal contamination and mediastinitis.

Esophageal perforation, regardless of cause or location, can lead to contamination of the retrovisceral space, with subsequent spread of cervical or thoracic suppuration to the posterior mediastinum [11]. Management of patients with esophageal perforation is determined by clinical status and radiological findings. Nonsurgical management is acceptable under certain conditions: the cavity around the perforation should be small and should drain back into the esophagus completely, and the patient should be asymptomatic without signs of local or systemic sepsis [12]. Interest in nonsurgical management is growing as endoscopy is more widely performed, more subtle perforations are detected because of increased awareness of complications of endoscopy and endoscopic procedures, and improved nutritional support and superior antibiotic therapy allow more aggressive nonoperative management [1, 13]. Nonsurgical management should not, however, be considered "conservative." A review of nearly 900 patients compiled from the literature [3] shows a mortality of almost 25% with "conservative" nonoperative management. Such a high mortality rate belies the modifier "conservative." The cornerstone of treatment of esophageal perforation remains surgical [2].

Primary repair of or diversion away from the esophageal perforation to eliminate ongoing mediastinal soilage is the key to surgical management. Primary repair of an esophageal perforation is aided by use of vascularized pedicle flaps, such as pleural flaps, and buttressing gastric fundal patches, both of which can aid in healing and help prevent leakage [11]. Patients in whom the esophageal perforation cannot be repaired should undergo esophageal diversion, proximal esophageal occlusion with decompression, or stenting across the perforation to eliminate ongoing mediastinal contamination, as well as wide drainage. Thorough drainage of the mediastinum is vital, although it should be viewed only as an adjunct to diversion or repair for control of ongoing contamination. Wide mediastinal drainage alone is inadequate, as shown by this review and others [3]. Mediastinal drainage is used to remove ongoing mediastinal soilage and to control ongoing mediastinal suppuration that occurs after repair or diversion is accomplished.

There has been some interest in extirpative treatment of esophageal perforations involving staged operations [8, 14], but the indications for such treatment appear to be limited [11], being directed primarily toward extensive esophageal devitalization. Gastrostomy tube decompression generally appears warranted to diminish possible gastroesophageal reflux and mediastinal soilage. Later, gastrostomy tube feedings may provide enteral nutritional support. Gastrostomy tube placement, however, has the potential to interfere with later reconstruction by altering the anterior gastric wall. A gastrostomy tube therefore must always be placed with the long-term care of the patient in mind.

In the patients of this review, mediastinal infection had two basic forms. In 3 patients, 2 of whom were not operated on because of "do not resuscitate" status, this infection was predominantly a diffuse suppurative process. The failure to drain the suppuration adequately resulted in overwhelming sepsis and death. In the remaining patients, the mediastinal infection took the form of a nonsuppurative necrotizing diffuse cellulitis. The degree of this latter type of mediastinal infection appears to be related to the length of delay in making the diagnosis, the extent of mediastinal contamination, and the adequacy of operative debridement and mediastinal drainage. Extensive and diffuse nonsuppurative necrotizing mediastinal cellulitis approaches mediastinal necrosis and imparts serious systemic effects. Prompt diagnosis and intervention, elimination of ongoing mediastinal contamination, mediastinal debridement, thorough mediastinal drainage, and aggressive antimicrobial therapy are the best methods to prevent development of nonsuppurative mediastinal necrosis and its complications.

Bacteriological evaluation of operative mediastinal specimens resulted in detection of polymicrobial infections in most patients in this review, although cultures of specimens from 21% of the patients yielded no bacteria. Aerobic bacteria and *Candida* were most commonly isolated. The number of bacterial species cultured at operation directly correlated to the time between perforation and exploration. Specimens from patients in whom the diagnosis of esophageal perforation was made more than 24 hours after perforation occurred were uniformly associated with polymicrobial infections. Two patients' cultures grew *Bacteroides fragilis*, a rather uncommon anaerobic mediastinal pathogen as it rarely occurs in normal human oral flora [15]. Infections involving untreated *Bacteroides* species can lead to impressive and serious bacteremia; approximately one third of the patients with *Bacteroides* bacteremia have septic shock [16]. Therefore, antimicrobial therapy initially must be broad and aggressive, protecting patients against gram-positive and gram-negative bacteria and aerobic and anaerobic bacteria. Later, antimicrobial therapy can be directed more specifically by bacteriological testing performed on operative samples, and antifungal therapy can be instituted if indicated.

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Can AIDS Be Prevented After Injury With Contaminated Instruments?

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Particulate matter comparable in size with that of human immunodeficiency virus was subcutaneously injected into experimental animals. Such matter remained at the inoculation site long enough to suggest the possibility that human immunodeficiency virus can be destroyed in loco before it invades the host's circulation. These findings may be useful in developing a method to prevent acquired immunodeficiency syndrome after accidental injury with human immunodeficiency virus–contaminated instruments.

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> I am in blood stepp'd in so far, that, should I wade no more, returning were as tedious as go o'er.
>
> *Macbeth*, Act III, Scene 4

Human immunodeficiency virus (HIV), also known as human lymphotropic virus III or lymphadenopathy-associated virus, is the proven cause of acquired immunodeficiency syndrome (AIDS). Presently there are thought to be 1.2 million persons infected with HIV, more than 57,000 cases of AIDS diagnosed, and about 33,000 deaths in the United States alone. These numbers are expected to multiply during the next few years [1].

Although HIV has been recovered from the saliva and tears of infected individuals, the disease is believed to propagate exclusively through sexual secretions and blood [2]. Those who are most likely to contact AIDS from HIV-infected blood are intravenous drug addicts sharing needles and patients who have received inadequately screened blood transfusions. To these, a smaller but nonetheless important group has been added: health workers, who because of their daily exposure to HIV-infected patients have a markedly increased potential for acquiring AIDS [1]. This fact is now being documented with increased frequency. Although some of these accidental infections occurred after exposure of abraded skin or mucous membranes to blood, most AIDS cases resulting from health-care–related accidents are caused by needle-sticks and by superficial cuts [3–16]. Although the risk of HIV transmission by such cuts and needle-sticks is relatively low, especially if compared with sharing of contaminated needles among drug addicts, the chance of such transmission is certainly great enough to cause deep concern among health care workers, especially surgeons, operating room nurses, and researchers. As of September 1988, 5.1% of the 61,929 adult cases of AIDS occurred among individuals employed in health care or clinical laboratory settings [8, 17]. Although 95% of these reported high-risk behavior, 5% (169 workers) had undetermined means of HIV acquisition, a much higher share than is found among people in other professions [18]. The number of confirmed cases in which seroconversion occurred after known contact with HIV-infected blood is also increasing [16]. Although a multiplicity of measures have been recommended to decrease the risk of such accidents, no single source conveys any practical information about measures to take if such an accident happens despite all precautions taken [1]. Currently, trials are underway to determine the possible usefulness of azidothymidine, an antiretroviral agent, in postexposure prophylaxis, but its value has not been proved. The only advice given in the latest relevant reports of Centers for Disease Control for surgeons or other health workers injured with contaminated needles or other instruments is that the person should have himself tested for AIDS in 6 weeks, refrain from sexual activity and seek counseling [8, 10].

Short of denial of care, a stance judged unethical by most members of our profession, the risk of AIDS to health care workers will not only continue to exist but, with the growth of number of HIV-positive patients, is expected to grow. Therefore, it is mandatory that treatment protocols be developed not only with regard to protecting ourselves and our associates by preventing injuries that may lead to AIDS but also with regard to methods of inactivating the HIV after inoculation. Those who seek to establish such protocols should consider that some features peculiar to accidental HIV infection of health professionals may be advantageously used in the prevention of AIDS: (1) The moment when the infection occurs is immediately known, (2) the injury that introduces the virus is usually limited in both extent and depth, and (3) the accident occurs in a well-controlled hospital environment. In addition, considering that HIV is known to be highly sensitive to some chemically and biologically active substances [2], a potential solution may be found by trying to destroy the virus while it still is confined to the site of injury.

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To study the feasibility of these considerations, experimental observations were made to study how particulate matter comparable in size with HIV is absorbed from the inoculation site and whether such matter remains confined to the site of entry long enough to be destroyed in loco before it invades the host's circulation.

Material and Methods

In a previous study (unpublished observations) we investigated the propagation of both water-soluble compounds such as iopamidol (Isoview, Squibb, New Brunswick, NJ), a radiographic contrast material, and fluorescein dye (Fluorescite, Taylor Pharmacological, Decatur, IL) as well as colloidal technetium suspension (Technecol, Mallinckrodt, St. Louis, MO) in the subcutis. Examining human volunteers and experimental animals, we found a wide range of differences in the amount of time these substances stayed within a 3-cm radius centered on their point of subcutaneous introduction; this time varied from a minimum of 1 minute 10 seconds to more than 18 minutes. Aqueous solutions left the area of introduction faster than colloid material. Furthermore, subcutaneous propagation was greatly accelerated if the material was introduced by injection rather than by inoculation through a knife-inflicted skin wound.

To establish a more acceptable model for the study of the passage of subcutaneously introduced HIV, we used a suspension of HIV-size polystyrene beads (PSB) (Seradyn Particle Technology Division, Dow Chemical, Indianapolis, IN) labeled with iodine 125. The diameter of the beads was 90 ± 8.6 nm (standard deviation), which closely approximates the 100-nm diameter of HIV. The PSB were iodinated with low-pH, medium-concentration $K^{125}I$ (New England Nuclear, Boston, MA) by diluting 100 μL of a 10% PSB suspension with 1.0 mL absolute ethanol, adding 50 μL of 0.05 mol/L ferric nitrate catalyst followed by 20 μL of $K^{125}I$ solution and allowing the reaction mixture to stand at room temperature for ten to 12 hours. Then 5 mL of ethanol was added and mixed, and the reaction mixture was centrifuged at 8,000 *g* for 10 minutes. The supernatant was removed and the ethanol wash was repeated twice. The labeled beads were then washed in a similar manner with 5 mL of 50% (vol/vol) ethanol and three times with deionized water containing sodium dodecyl sulfonate, 100 mg/100 mL. These last washes were centrifuged at 70,000 *g* to separate the beads. Even at this speed, the separation was not quantitative. The supernatant was carefully pipetted off immediately because of spontaneous resuspension of particles when the centrifuge stopped. Complete redispersion of the particles required ultrasonic treatment for one to two minutes. Each preparation was diluted to contain approximately 1.3×10^5 cpm per 10 μL just before injection.

Twenty-two mixed-breed dogs were used in the study with PSB, all maintained and treated according to the "Guide for the Care and Use of Laboratory Animals" (NIH publication No. 85-23, revised 1985). Each animal was anesthetized with sodium pentobarbital during the experiment and killed at its completion with T-61 euthanizing solution (Hoechst-Roussel Agri-Vet, Somerville, NJ).

Each animal was prepared by exposing a left or right femoral vein, administering heparin, and cannulating the vein. A subdermal lymph duct was made visible by injecting 1 mL of Evans blue dye subcutaneously at knee level. Lymph samples were collected on a Weck-Cel surgical spear (Edward Weck and Co, Inc, Research Triangle Park, NC). Samples of blood and lymph were then obtained as a control. One hundred microliters of the ^{125}I-labeled PSB was injected subcutaneously with light pressure 10 cm distal to the site of cannulation. Blood samples (4 mL) were withdrawn from the vein cannula every 20 seconds for the first four minutes, at one-minute intervals between four and ten minutes, and at two-minute intervals between ten and 20 minutes. Lymph samples were collected every minute for the first ten minutes and every two minutes between ten and 20 minutes. In six experiments, after the 20-minute sample was obtained, the injection site was gently massaged for the first 30 seconds of each minute; blood and lymph samples were taken at the end of each minute for an additional five minutes. The tubes were capped, and the radioactivity of each sample was measured in a Multiprias gamma counter (Packard Nuclear, Chicago, IL).

Results

The efficiency of labeling was 10% of the added radioactivity bound to the beads. For injection, the radioactivity of the bead suspension was adjusted so that the 100 μL injected contained 1.3×10^6 cpm.

Precise quantitative data were difficult to obtain for the labeling procedure because of the incomplete separation of particles from the wash liquid. This difficulty was overcome by the recognition that incomplete removal of ^{125}I physically absorbed in the PSB produced a sharp peak that appeared very early (less than one minute) in the femoral vein blood samples and that three washes with 100% ethanol eliminated all traces of the early peak without any effect on the later peak caused by the radioactive polystyrene particles.

Figure 1 shows the transfer time for ^{125}I-labeled PSB from the subcutis to the femoral vein blood and to the lymph. The average transfer time for radioactive iodide into the blood was 1 minute 20 seconds ± 16 seconds. The average transfer time for the HIV-size beads into the blood was 4 minutes 6 seconds ± 49 seconds ($p < 0.001$) and 7 minutes 45 seconds ± 2 minutes 19 seconds into lymph samples.

Comment

From our observations, we conclude that (1) particulate matter of the size of HIV introduced into the subcutis will remain confined to the injection area for at least one minute and probably longer; (2) particulate matter of HIV size leaves the extremity by the large veins rather than through lymph channels; and (3) development of retrovirus infection after accidental inoculation with virus or virus-infected blood might be prevented by local mea-

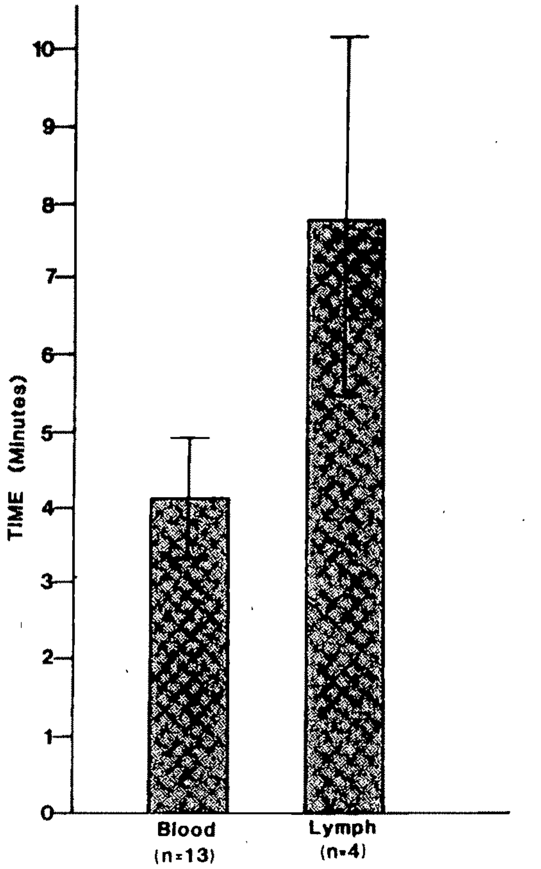

Fig 1. Time in minutes from subcutaneous injection of ^{125}I-labeled polystyrene beads to the appearance of radioactivity in femoral vein blood or subdermal lymph duct fluid (mean ± standard error of the mean).

sures. The validity of this postulate needs to be proved in animal model studies, which are ongoing in our laboratory.

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Distal Aortic Pressure During Coarctation Operation

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Mean distal aortic pressure (DAP) was monitored continuously during operative repair of coarctation of the aorta in 67 children more than 1 year of age between 1982 and 1987. At initial test clamping a DAP of 45 mm Hg or more was considered adequate. In 42 patients (group A), DAP exceeded 45 mm Hg and the operation proceeded. In 25 patients (group B) the DAP was less than 45 mm Hg, and adjustments were made to the position of the proximal and distal clamps and in the use of hypotensive agents. After these adjustments in group B, DAP rose from 34.7 to 50.6 mm Hg ($p < 0.001$), achieving the desired level in all but 5 patients, who required temporary shunts to support the distal circulation. The adjustments were as follows: the left subclavian artery was open in 28% of patients before and 60% after ($p < 0.02$); no more than one pair of intercostal arteries was clamped in 64% before and 88% after ($p < 0.05$); and no hypotensive agents were used in 56% before and 80% after ($p = 0.07$). During the first ten minutes of cross-clamping the DAP rose by 5.5 mm Hg ($p < 0.01$, n = 52). It is concluded that continuous monitoring of DAP contributed to the operative management of these patients by indicating when adjustments in the position of the clamps or in the use of hypotensive agents were needed. The use of hypotensive agents should be reduced to achieve a higher DAP.

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Paraplegia complicates repair of coarctation of the aorta with a reported incidence of 1 in 250 patients [1, 2]. Two possible mechanisms are interruption of a critical segmental supply to the anterior spinal artery and cord [3, 4] and inadequate distal aortic flow or pressure during the period of cross-clamping [4–7]. In 1971, Hughes and Reemtsma [8] proposed that the mean distal aortic pressure (DAP) should be measured by formal manometry at the time of clamping. If DAP was less than 50 mm Hg then atriofemoral bypass should be used to support the distal circulation [8]. In 1982, we adopted a policy of manometric measurement of DAP throughout the entire operation, and our findings demonstrate that this measurement contributed to the operative management of patients undergoing coarctation operation.

Material and Methods

Between 1982 and 1987, 133 patients underwent surgical repair of coarctation of the aorta. The age distribution of 67 patients who were more than 1 year of age and were the subject of this study is shown in Table 1. Sixty operations were for primary coarctation, and seven were for a recurrence. Forty-eight patients underwent resection and end-to-end anastomosis and 17 patients had patch aortoplasty; 1 patient had a Dacron bypass graft and 1 had subclavian flap aortoplasty.

After induction of anesthesia, arterial cannulas were placed in both the right upper limb and one lower limb. Anesthesia was maintained with nitrous oxide-oxygen and a relaxant technique supplemented with opiates and either halothane or enflurane. After thoracotomy, the aorta was mobilized in preparation for aortic cross-clamping. A combination of inhalational and intravenous agents (halothane, enflurane, and sodium nitroprusside) was used in the conventional way with the objectives of controlling the proximal pressure and maintaining an adequate distal aortic pressure.

When mobilization was complete, test clamping of the aorta was performed. The clamps were initially placed at a position judged to be comfortable for the surgeon. A mean DAP of 45 mm Hg or greater at initial test clamping was accepted as adequate. This figure was adopted, rather than the 50 mm Hg advised by others [8], as a preliminary study suggested that the DAP rose during the first ten minutes of aortic cross-clamping. The operation proceeded if the mean DAP was found to be more than 45 mm Hg. If it was less than 45 mm Hg, then attempts were made to increase the distal pressure by: (1) adjusting the position of the proximal clamp to open the left subclavian artery; (2) adjusting the position of the distal clamp to open the maximum number of intercostal arteries; and (3) raising the proximal pressure either by reducing or stopping agents that might lower that pressure.

If after these adjustments the DAP was still less than 45 mm Hg, the distal circulation was supported either by a temporary shunt from the distal aortic arch to the descending thoracic aorta or by partial left heart bypass.

The statistical analysis included the use of Student's *t* test, paired *t* test, contingency table, χ^2 analysis, and Fisher's exact test.

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Table 1. Age Distribution of 67 Patients Older Than 1 Year

Age (yr)	No. of Patients
1–5	36
5–10	13
10–15	8
15–20	4
>20	6

Results

There were no deaths or spinal cord injuries in these patients. Forty-two of the 67 patients achieved the desired level of DAP on test clamping, and the operation proceeded forthwith (group A). Twenty-five patients initially had a DAP less than 45 mm Hg on test clamping (group B). They underwent one or more of the adjustments, and as a result, 20 of the 25 patients succeeded in attaining the desired distal pressure. It was necessary to support the distal circulation in the remaining 5 patients.

Before the aorta was test-clamped, there was no significant difference between groups A and B in proximal or distal aortic pressures (Table 2). On test clamping, the proximal aortic pressure rose to a similar extent in both groups A and B. The DAP fell to a mean of 58.6 mm Hg in group A and to the significantly lower mean of 34.7 mm Hg in group B ($p < 0.01$). After adjustments had been made to the position of the clamps or in the use of hypotensive agents, or both, in the 25 group B patients (Table 3), there was a significant rise in both the proximal systolic pressure (rising from 150 to 163.7 mm Hg, $p < 0.02$) and the distal mean pressure (rising from 34.7 to 50.6 mm Hg, $p < 0.01$), so that 20 patients achieved an adequate distal pressure. The remaining 5 patients had the distal circulation supported by a temporary aortoaortic shunt in 4 patients and by left atriofemoral bypass in 1 patient.

At the time of test clamping, there was no statistically significant difference in management between the two groups for the following variables (Table 4): (1) the position of the proximal clamp relative to the left subclavian artery; (2) the position of the distal clamps resulting in not more than one pair of intercostals being obstructed; and (3) the percentage of patients in whom no hypotensive agents were used.

Table 2. Aortic Pressure Before and During Test Clamping

	Aortic Pressure (mm Hg)	
Time	Proximal	Distal
Before clamping		
Group A (n = 42)	135.0 ± 18.2	75.2 ± 13.7
Group B (n = 25)	133.2 ± 15.6[a]	70.9 ± 13[a]
Test clamping		
Group A (n = 42)	146.0 ± 18.8	58.6 ± 12.4
Group B (n = 25)	150.5 ± 27.3[a]	34.7 ± 5.8[b]

[a] Not significantly different from group A. [b] Significance: $p < 0.01$ versus group A.

Table 3. Aortic Pressure in Group B After Adjustments

	Aortic Pressure (mm Hg)	
Time	Proximal	Distal
Before adjustment	150.5 ± 27.3	34.7 ± 5.8
After adjustment	163.7 ± 32.6[a]	50.6 ± 7.3[b]

[a] Significance: $p < 0.02$ versus before adjustment. [b] Significance: $p < 0.001$ versus before adjustment.

Significant changes were made in group B patients: the left subclavian artery was open in 28% of the patients before adjustment and in 60% after ($p < 0.02$). No more than one pair of intercostal arteries was clamped in 64% of patients before adjustment and in 88% after ($p < 0.05$). The percentage of patients in whom no hypotensive agents were used rose from 56% to 80% ($p = 0.07$). Fifty-nine and a half percent of patients received no hypotensive agent during the period of cross-clamping. The reduced use of hypotensive agents led to a higher proximal systolic aortic pressure than had been accepted previously (see Table 3). Before clamping, 2 patients had proximal systolic pressure greater than 160 mm Hg; after clamping, 19 patients had pressures between 160 and 180 mm Hg, and 3 patients exceeded 200 mm Hg temporarily, whereupon hypotensive agents were introduced (Table 5).

There was a rise in the mean DAP during the first ten minutes of aortic cross-clamping from 53.3 to 58.8 mm Hg (n = 52, $p < 0.01$).

Comment

Paraplegia complicating coarctation operations remains one of the most feared problems in cardiac surgery. It is widely accepted that the injury is due to inadequate blood flow to the spinal cord. Two mechanisms are thought to lead to spinal cord ischemia and paraplegia: inadequate distal aortic flow or pressure and the interruption of a critical segmental arterial supply to the anterior spinal artery and the spinal cord. Increased cerebrospinal fluid pressure is a possible third factor that may decrease perfusion pressure across the cord, but conflicting results have been reported [6, 7, 9–11]. This study addressed the first factor, the adequacy of the DAP.

It is not known what level of DAP is either adequate or inadequate during aortic cross-clamping in patients with

Table 4. Management at Initial Clamping

Group	LSA open	Not More Than 1 Pair of Intercostals Clamped	No Hypotensive Agents
A (n = 42)	33.3%	59.5%	42.9%
B (n = 25)	28%[a]	64%[a]	56.0%[a]

[a] Not significantly different from group A.

LSA = left subclavian artery.

Table 5. Number of Patients at Each Level of Proximal Aortic Pressure During Aortic Clamping

Time	Proximal Systolic Pressure (mm Hg)			
	<160	160–179	180–199	200>
Before clamping	65	2	0	0
After clamping	45	19	0	3

coarctation of the aorta. The adequacy of spinal cord blood flow during cross-clamping has been evaluated using somatosensory evoked potentials [12, 13] and measurement of either distal organ blood flow or oxygen tension [5–7]. Many of these studies have been in the absence of coarctation and collateral formation.

The New York University group [12, 14] has used somatosensory evoked potentials in both clinical and experimental settings to accurately assess cord ischemia during aortic cross-clamping; however, for the most part, this was not in a coarctation model. They have reported 33 patients undergoing thoracic aortic cross-clamping, but only 3 were patients with coarctation [12]. In another report on 103 patients with coarctation [14], 5 patients had evoked potentials measured, each of whom had a mean DAP during aortic clamping of more than 60 mm Hg; the evoked potential remained unchanged and no paraplegia occurred.

Experimental evidence of the relationship between distal aortic pressure and spinal cord blood flow comes from an Alabama study [5] in a noncoarctation model: aortic cross-clamping in 11 dogs led to a significant drop in mean arterial pressure below the clamp, with an associated decrease in renal and spinal cord blood flow. The introduction of a sodium nitroprusside infusion further decreased the distal mean aortic pressure and the renal blood flow. This is the "nitroprusside steal" phenomenon [9], in which there is shunting away from the relatively high-resistance spinal cord collaterals to the proximal low-resistance arterial bed. There was a strong association between renal blood flow and mean DAP, and the tentative conclusion was reached that blood flow in the organ below the clamp was pressure dependent.

In another noncoarctation model, the Hanover group [6, 7] experimentally identified decreased spinal cord tissue oxygen tension as the primary reason for spinal cord damage. This was in turn related to pressure-dependent blood flow to the distal cord. In their series, those pigs with a critical drop in oxygen tension of the spinal cord had a DAP of 15 to 20 mm Hg.

The Toronto Children's Hospital Group [13] studied the occurrence of reversible spinal cord ischemia demonstrated by the loss or diminution of somatosensory evoked potentials in 38 patients undergoing aortic cross-clamping for repair of coarctation of the aorta. In 31 patients, the DAP was also measured and was 32.4 ± 2.4 mm Hg. A lower DAP was identified as a risk factor for reversible spinal cord ischemia. In turn, the DAP was reduced by the use of vasodilators and a low arterial carbon dioxide tension.

Our study commenced in 1982 before publication of the above reports, and we followed the lead of Hughes and Reemtsa [8] in measuring the DAP. However, we chose continuous intraarterial monitoring rather than a single-needle measurement on clamping. This enabled us to observe both proximal and distal aortic pressures before cross-clamping, at test clamping, after clamp adjustments, during clamping, and afterwards. During a pilot study, the distal mean pressure rose during the first ten minutes after clamping, and this has been confirmed in the present study. The pressure then tended to plateau in the majority of patients, but in some it continued to increase. This rise that follows clamping may be taken into account when judging the adequacy of DAP at test clamping. Thus, we accepted 45 mm Hg as a satisfactory initial measurement rather than the previously recommended figure of 50 mm Hg.

Forty-two of our patients (group A) had a satisfactory distal pressure when the clamps were first applied, and their operation proceeded; the remaining 25 patients (37.3%; group B) required adjustments. The initial placement of the clamps was in such a way that the surgeon found it comfortable to perform the operation. Judicious readjustment of the clamps to allow left subclavian artery flow or to allow more intercostal arteries to remain open was of importance in most patients in group B to achieve a satisfactory DAP. It was a maneuver without major disadvantage as the operative procedure was always technically feasible, and its favorable effects could be observed on both proximal and distal aortic pressure.

Agents that lower proximal arterial pressure have traditionally played an important role in the intraoperative management of coarctation operations. Early in the study, it became evident that the lowering of proximal aortic pressure had the deleterious effect of also lowering mean DAP. The use of halothane, enflurane, sodium nitroprusside, or labetalol to lower the proximal aortic pressure to 150 mm Hg or less contributed to an unacceptably low level of DAP in a number of patients in group B. To achieve a satisfactory DAP, intraoperative management was altered. After adjustment to the position of the clamps and withdrawal of hypotensive agents in the patients in group B proximal pressure rose significantly as well as distal pressure. Manipulation of the clamps would be expected to reduce proximal pressure. This suggests that the reduced use of hypotensive agents led to a physiologically important increase in the proximal pressure and to a resultant rise in distal pressure, presumably owing to increased pressure-dependent flow through high-resistance vessels. The evolution of intraoperative management led to a significantly decreased use of blood-pressure–lowering agents. During clamping, no hypotensive agents were given to 59.5% of the patients.

In a recent report from Birmingham, United Kingdom [15], the advice of Hughes and Reemtsma [8] was followed strictly, so that in a series of 47 patients more than 1 year of age, 22 patients (47%) were found to have a DAP below 50 mm Hg and proceeded to have left heart bypass. There was no paraplegia but 1 patient may have had an air embolism. Our experience would suggest that modification of operative management would have enabled bypass to be avoided in the majority of the these cases.

The reduced use of hypotensive agents led to a higher proximal systolic pressure than had been accepted previously. A compromise was sought between a proximal pressure that was excessively high and one that was excessively low, which might in turn reduce the distal pressure to an unacceptable level. We now aim to maintain a systolic pressure below 180 mm Hg during the period of clamping, and measures are instituted to lower the pressure when this figure is exceeded. Arguments against this level of pressure include the potential complications of cerebral hemorrhage and acute left ventricular failure. Neither of these complications occurred in this series, and it is likely that young children with unrelieved coarctation will achieve these levels of arterial pressure when crying or undertaking exercise. We do, however, acknowledge this theoretical disadvantage of a policy that employs a higher proximal pressure.

We agree with the assertion [5, 13] that sodium nitroprusside infusion should be used with caution during clamping because it may cause a reduction in the distal mean pressure with a consequent decrease in tissue blood flow below the clamp. This conclusion may be extended in the clinical setting to any agent that lowers the proximal pressure.

By using the simple measures of readjustment of the position of the clamps and reduced use of hypotensive agents, more than 90% of patients in this study attained a distal mean aortic pressure of 50 mm Hg or more.

From this study, we draw the following conclusions.

1. Measurement of DAP contributed positively to the operative management of patients undergoing operation for coarctation by identifying those with inadequate distal pressure on clamping and by demonstrating the results of adjustments to the position of the clamps or in the use of hypotensive agents.
2. Sixty-two of 67 patients achieved an acceptable mean DAP. In 20 this was achieved by changes in intraoperative management after measurement of an inadequate distal pressure on test clamping. In 5 of 67 patients support of the distal circulation was necessary.
3. Distal aortic pressure rose during the first ten minutes of aortic cross-clamping.
4. A higher proximal aortic pressure permitted a higher distal pressure. We advocate where necessary a reduction in the use of hypotensive agents during the period of cross-clamping to achieve a satisfactory mean DAP.

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Operative Treatment of *Fusarium* Fungal Infection of the Lung

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Systemic fungal infections with *Fusarium* occur predominantly in immunocompromised patients and are usually fatal. We report a patient with acute lymphocytic leukemia and fusariosis involving the skin and lungs. This patient underwent antifungal chemotherapy and bilateral pulmonary resections. She subsequently had successful bone marrow transplantation. The results of this treatment suggest that this aggressive management of pulmonary fusariosis offers the best chance of survival.

(Ann Thorac Surg 1990;49:991–2)

Fusarium is a genus of fungi first reported to produce systemic infection in humans in 1973 [1]. Since then, 28 additional cases have been described [2]. All affected patients have been immunosuppressed, and most died despite vigorous antifungal chemotherapy. Lung infection with *Fusarium* carries a particularly grim prognosis. We have recently treated a patient with acute lymphocytic leukemia and *Fusarium* infection of both lungs. The patient received antifungal chemotherapy followed by bilateral pulmonary resections. She underwent successful bone marrow transplantation and remains free of infection.

The patient is a 15-year-old Brazilian girl in whom acute lymphocytic leukemia developed in 1985. She received chemotherapy for 3 years but had a bone marrow relapse 6 months after completion of chemotherapy. Attempts at reinduction chemotherapy failed to achieve a second remission. Arrangements were made for transfer to the University of Iowa Hospitals and Clinics for possible bone marrow transplantation. In December 1988, before the patient was transferred to our institution, fever and productive cough developed. She was examined at a hospital in Sao Paulo, Brazil, and received intravenous antibiotics while awaiting transfer to the University of Iowa Hospital.

Upon admission to our institution on January 13, 1989, the patient was cushingoid with an oral temperature of 39.3°C. There was no lymphadenopathy and the lungs were clear. The left forearm had a 2 × 2-cm erythematous, tender, raised lesion. A chest roentgenogram showed a right upper lobe infiltrate and a left lower lobe nodular density (Fig 1). The patient underwent punch biopsy of the forearm lesion, which demonstrated fungal elements consistent with *Fusarium*. Cultures confirmed *Fusarium* infection. She received multiple antibiotics including amphotericin B throughout this time and became afebrile on January 19. Serial bone marrow examinations indicated the patient had entered bone marrow remission, making the suspected fusariosis the greater immediate concern. Computed chest tomography (Fig 2) confirmed the pulmonary distribution of the lesions and excluded abdominal and intracranial involvement. The patient was referred for pulmonary resection.

On February 13, the patient underwent posterior segmentectomy of the right upper lobe. This segment showed destruction and cavitation, but the pathology did not traverse the segmental boundaries. Although tissue cultures were negative, microscopic examination showed fungal forms consistent with *Fusarium*. On March 10, the patient underwent wedge resection of two small nodular densities from the left lower lobe. The pathological diagnosis again was *Fusarium*. The patient made an uneventful recovery from these operations.

Administration of amphotericin B was continued throughout the pretransplantation period, but antileukemic therapy was withheld to minimize immunosuppression. Neutrophil counts remained adequate ($>5 \times 10^8$/L) during this time. On April 7, the patient received a bone marrow transplant from her HLA-identical sister. Prompt engraftment occurred, and she was discharged from the hospital on May 16, 1989, to be followed up as an outpatient. The frequency of amphotericin administration was gradually tapered, and the medication was discontinued 100 days after transplantation. There has been no evidence of recurrent fungal disease during the follow-up period.

Comment

The genus *Fusarium* includes a number of fungal species that are found in the soil and are important plant pathogens [3]. Species identification is difficult because these organisms may change their most salient features, morphology and color, in vitro [4]. *Fusarium* may be morphologically indistinguishable from *Aspergillus*, which also displays septate branching hyphae and shares a propensity to vascular invasion [5]. *Fusarium* may resemble species of *Petrielidium* and *Cephalosporium* [3, 6]. Concurrent infections with bacteria or other fungi are also common [4].

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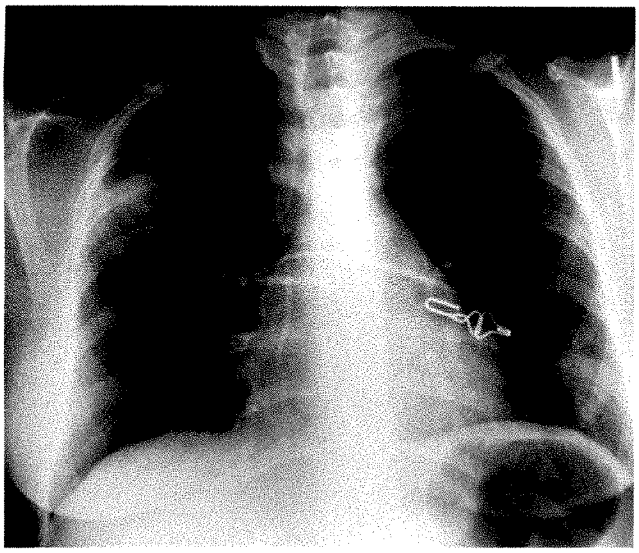

Fig 1. Preoperative chest roentgenogram demonstrating infiltrates in the right upper and left lower lobes.

Superficial *Fusarium* infections of the skin, nails, and cornea are frequent, and colonization and infection of skin ulcers [7] or burn wounds [8] may occur. Skin biopsy in the appropriate clinical setting may permit early diagnosis of *Fusarium*. Fusariosis involving at least two separate organs was first reported in 1973 [1]. The latest review of systemic fusariosis includes 29 patients, 26 of whom were treated for hematologic malignancies [2].

Survival after disseminated fusariosis is rare. Treatment of *Fusarium* has relied primarily on amphotericin B, with 5-fluorocytosine, ketoconazole, and miconazole sometimes used to achieve a synergistic antifungal effect. In vitro, *Fusarium* is usually resistant to ketoconazole [6] and 5-fluorocytosine [2, 6, 8]. Sensitivities to amphotericin B and miconazole are more varied, with half of isolates sensitive and half resistant [2, 4, 6, 8, 9]. Antifungal chemotherapy is most successful when infection is limited to the skin or only blood cultures are positive.

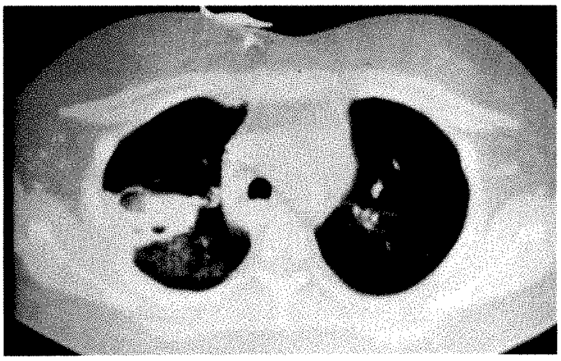

Fig 2. Computed tomogram of the chest demonstrating consolidation of the posterior segment of the right upper lobe.

The lung is the third most common site of *Fusarium* infection, following skin and blood. Pulmonary involvement occurred in 12 of the 29 recently reviewed cases [2]. All 12 patients with pulmonary fusariosis died of the disease [2, 3–6, 8]. We believe the successful outcome of our patient is in part attributable to operative treatment of her pulmonary lesions. Bone marrow transplantation would have been prohibitively dangerous in the presence of any residual infection.

This patient was fortunate that the extent of her pulmonary infection was relatively limited. It is unlikely that all patients with *Fusarium* infections of the lungs can be successfully treated with surgical excision. The distribution of this patient's disease made her an excellent candidate for conservative resection. There would appear to be no advantage to wider resection, ie, lobectomy, although this may be required in cases of more extensive disease. We believe that surgical resection in pulmonary fusariosis merits further study in efforts to define curative treatment for this life-threatening infection.

Addendum

After the acceptance of the manuscript, cytomegalovirus pneumonia, a common complication of bone marrow transplantation, developed in this patient. This diagnosis was confirmed by open lung biopsy. Microscopic inspection as well as cultures of the lung tissue did not demonstrate any recurrent fungal infection. All efforts to treat the pneumonia were unsuccessful, and the patient died on February 22, 1990. Permission for an autopsy was not granted.

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Open Fixation of Flail Chest After Blunt Trauma

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Progressive left chest volume loss developed in a patient with severe flail chest despite reasonable oxygenation without intubation. Because of this chest volume loss, pain, and shortness of breath, she underwent open chest wall repair using multiple metallic struts. Rapid recovery ensued, despite a perforated duodenal ulcer on postoperative day 1. Benefits of open fixation of severe flail chest are clearly demonstrated and should be considered instead of prolonged ventilation or supportive care alone for select patients.

(Ann Thorac Surg 1990;49:993–5)

The early management of flail chest remains controversial. Because most patients may be managed conservatively, early operative fixation is rarely considered. The insidious progression of chest wall deformity and loss of lung function is demonstrated in the present case, as are the benefits of early operative fixation.

A 66-year-old woman sustained blunt left chest trauma in a motor vehicle accident. On arrival, decreased breath sounds and tenderness along her medial left scapular border and laterally along her entire upper chest wall were found. Moderate flail chest was demonstrated lateral and posterior to the anterior axillary line. A chest roentgenogram (Figs 1, 2) showed fractures of left ribs 2 through 7. Arterial blood gas on oxygen showed an oxygen tension of 131 mm Hg and a carbon dioxide tension of 42 mm Hg. A left chest tube was inserted because of a pneumothorax.

Despite analgesics, she continued to have severe pain on motion but minimal pain at rest. Progressive tachypnea (28 to 32 breaths per minute) despite administration of oxygen, fever, and progressive left lung volume loss (Fig 3) ensued. Although comfortable at rest, she would become markedly dyspneic with minimal exertion such as turning in bed or slow ambulation. Her oxygen tension when off oxygen ranged between 35 and 45 mm Hg. When examined, the chest wall was palpably unstable anteriorly, with a nonmovable, palpable step-off just medial to the scapula posteriorly. Operative reduction and fixation were recommended over other possible modalities (continued conservative management or intubation and mechanical ventilation).

At thoracotomy, ribs 2 through 8 were found to be fractured and the posterior portions of the fractured segments were tightly impacted under the medial surfaces of the rib fracture edges. Fracture reduction required entering the pleural space and considerable force to distract the impacted, displaced segment, which was bound down by early fibrosis and residual muscle spasm. Three Adkin struts (V. Mueller Company, Chicago, IL) were wired to the outer surface of the ribs using No. 5 stainless steel wires, bridging the fractures anteriorly and posteriorly to stabilize the chest wall. The flail segment was subsequently anchored to the struts, suspending it in the proper position.

The patient was extubated the next morning and complained of minimal chest pain in the operative area but had improved ability to take a deep breath. A follow-up roentgenogram later that afternoon demonstrated gross free air under the diaphragm; repair of a perforated duodenal ulcer was carried out uneventfully. She continued to do well and was reextubated the next morning. Early ambulation was easily accomplished, and she was discharged 13 days postoperatively. At follow-up visit (Fig 4), she reported no pain related to the struts. She is doing well 1 year after operation with her vital capacity 85% of predicted and a forced expiratory volume 115% of predicted.

Comment

Open fixation of flail chest is an uncommon operation, except when the chest is entered for other reasons. Mandatory mechanical ventilation of flail chest injuries has been replaced by selective intubation and mechanical ventilation only if patients are unable to support ventilation. Although this patient was able to support ventilation at all times, she was severely restricted in her ability to move because of pain and respiratory compromise. The initial severity of her injury was probably underestimated owing to the posterior, subscapular location of her flail chest wall segment, which tends to be less visible. Flail areas located under the scapular muscles, although better tolerated than in the more mobile anterior chest, can still lead to insidious respiratory compromise and progressive deformity. The initial blood gases suggested the need for intervention, and operation was selected so that the displaced fracture could be properly reduced. There is little doubt that this woman was helped by operation as shown by her immediately improved breathing ability, by radiographic resolution of her severe volume loss, and by her ability to withstand a major abdominal complication and operation within 24 hours.

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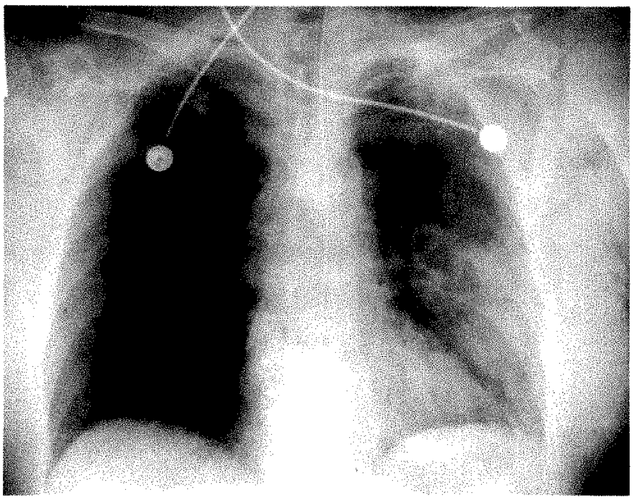

Fig 1. Admission chest roentgenogram shows multiple left rib fractures and left pulmonary contusion.

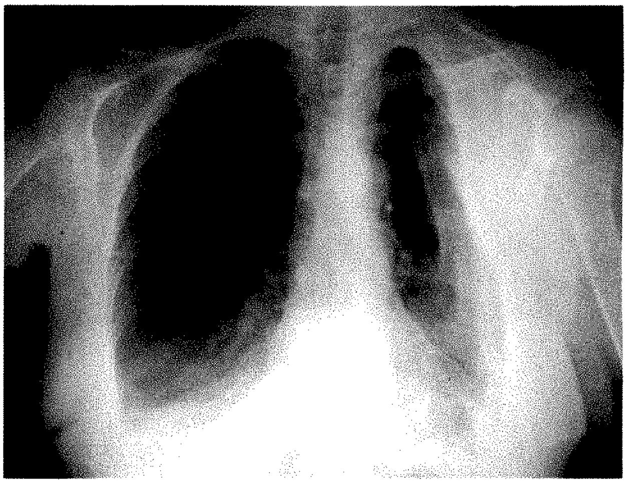

Fig 3. Chest roentgenogram several days after admission showing progressive volume loss in the left chest and progressive rib cage deformity.

The early operative stabilization of flail chest has been previously advocated both directly and indirectly [1–3]. Not only does it shorten ventilator time, but it may prevent considerable deformity and reduction in pulmonary function studies that can accompany randomly healed flail chest injuries. In addition, chronic (as well as acute) pain may be alleviated by proper operative stabilization as it was in this case.

Generally, the accepted indications for open fixation of flail chest are gross instability of a large segment of chest wall, other thoracic operation that allows for the simultaneous repair of a flail segment, or severe, unremitting pain related directly to the fractures. No randomized trial of open fixation versus prolonged conservative management or mechanical ventilation has been carried out, and

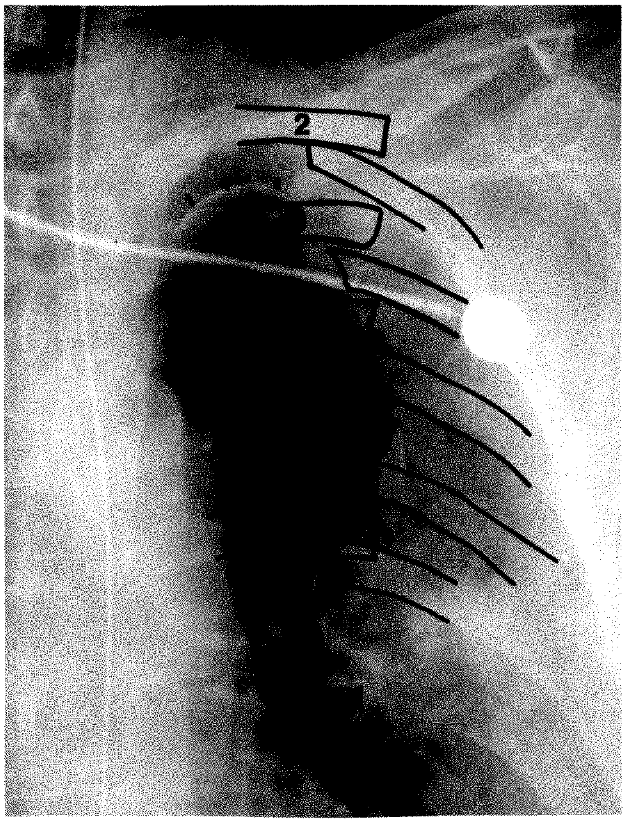

Fig 2. Numerals denote rib numbers and rib segments are highlighted to demonstrate fracture anatomy and displacement. Arrow *points to a poorly seen fracture in rib 7.*

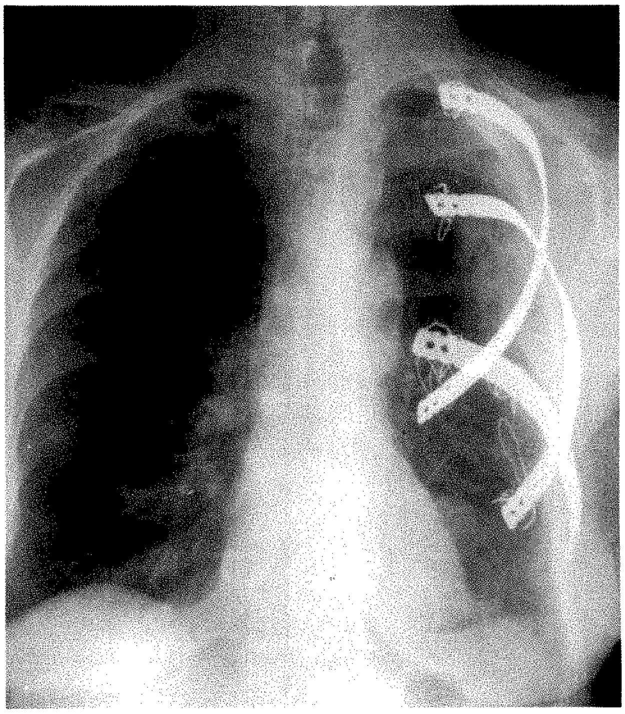

Fig 4. Posteroanterior chest roentgenogram 1 month postoperatively demonstrating position of struts and resolution of left chest volume loss.

such a study could add considerably to our understanding of this disease.

The surgical procedure is quite easy and does not necessarily require entering the pleura. Wires may be passed subperiosteally around the adjacent ribs using a subperiosteal tunnel. A variety of struts have been described [1] in addition to those used here. These can be bent manually or by using a mechanical plate bender to almost any curvature desired.

In summary, an example of early operative fixation of flail chest is described. In cases involving gross instability, chronic pain, or a progressively deteriorating clinical picture, operative fixation should be considered early on. Potential advantages of this technique include shortened hospitalization, reduced duration and need for mechanical ventilation, improved respiratory reserve, and decreased likelihood of clinically significant postoperative respiratory dysfunction and skeletal deformity.

I thank Kathy Pommer for secretarial support.

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Pleural Chondrosarcoma

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Thoracic extraskeletal chondrosarcomas are uncommon tumors. A case of primary chondrosarcoma of the pleura is presented. Complete surgical resection without adjuvant therapy, even in the presence of pleural seeding, appears to offer a favorable long-term prognosis.

(Ann Thorac Surg 1990;49:996–7)

Extraskeletal chondrosarcomas are uncommon tumors. This report describes a case of primary chondrosarcoma of the pleura.

A 70-year-old asymptomatic woman underwent routine physical examination in December 1975. Her medical history included a total abdominal hysterectomy for leiomyomata of the uterus in 1945. Physical examination was normal, but chest roentgenogram demonstrated a mass in the left posterolateral paratracheal area (Fig 1). Barium esophagogram and tomograms of the chest demonstrated this lesion to be located in the posterior mediastinum at the level of the aortic arch. There was extrinsic displacement of the esophagus to the right but no visceral or osseous invasion. Results of routine laboratory studies and radioisotope scans were normal. Thoracic aortic and superior vena caval angiograms demonstrated no invasion. Fiberoptic bronchoscopy was normal, and bronchial washings were negative for neoplastic disease. Cervical mediastinoscopy demonstrated a firm mass palpable posteriorly within the pleural space at the level of the fourth thoracic vertebra. A cutting-needle biopsy specimen was consistent with "cartilage."

A left thoracotomy was performed on April 14, 1976. Multiple smooth, firm, lobulated lesions were found attached to both parietal and visceral pleural surfaces. The largest lesion was based on the parietal pleura posteriorly at the level of the proximal descending aorta and measured 8 × 5 × 5 cm (Fig 2). It could be moved with the pleura and had no other fixation. A small portion of adjacent left upper lobe was adherent to it. A second lesion measuring 3 × 3 cm was attached to the parietal pleura on the dome of the left hemidiaphragm. Several other smaller lesions were noted on both the visceral and parietal pleural surfaces. All of these lesions, together with their pleural bases and any area of adjacent adherent lung, were excised.

Examination of the specimens by the Pathology Department of Walter Reed Army Medical Center and by the Bone, Soft Tissue, and Pulmonary Branch of the Armed Forces Institute of Pathology indicated the lesions to be chondrosarcomas (Fig 3). No evidence of parenchymal, osseous, or metastatic origin could be identified. Excised lesion demonstrated microscopic invasion of the adjacent lung, diaphragm, or soft tissue.

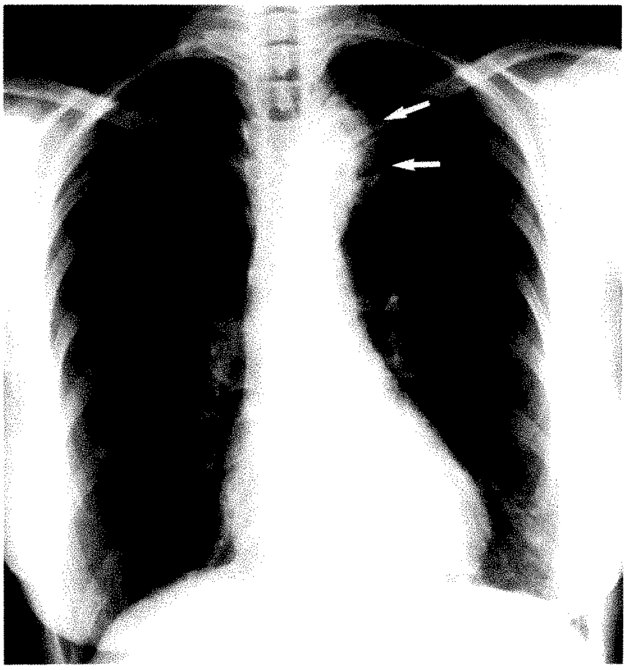

Fig 1. Preoperative chest roentgenogram demonstrating left paratracheal mass (arrows).

The patient made an uneventful recovery. No adjuvant therapy was recommended. Serial examinations including chest roentgenograms have failed to demonstrate any evidence of recurrence, and she is currently alive 13½ years after diagnosis.

Comment

Primary extraskeletal chondrosarcomas have been reported in many sites, but they are uncommon and usually arise from the deep soft tissue of the extremities, especially of the thigh and popliteal fossa. Such neoplasms usually occur in adults over 35 years of age and have a slight male preponderance.

Extraskeletal chondrosarcomas of the thorax are rare. A total of 17 adequately documented pulmonary chondrosarcomas have been reported in the literature [1–4], six occurring in the trachea and 11 in the peripheral bronchi and lung. Prognosis was good when the tumor was located centrally and could be totally excised. Peripheral tumors were more invasive, and most patients died

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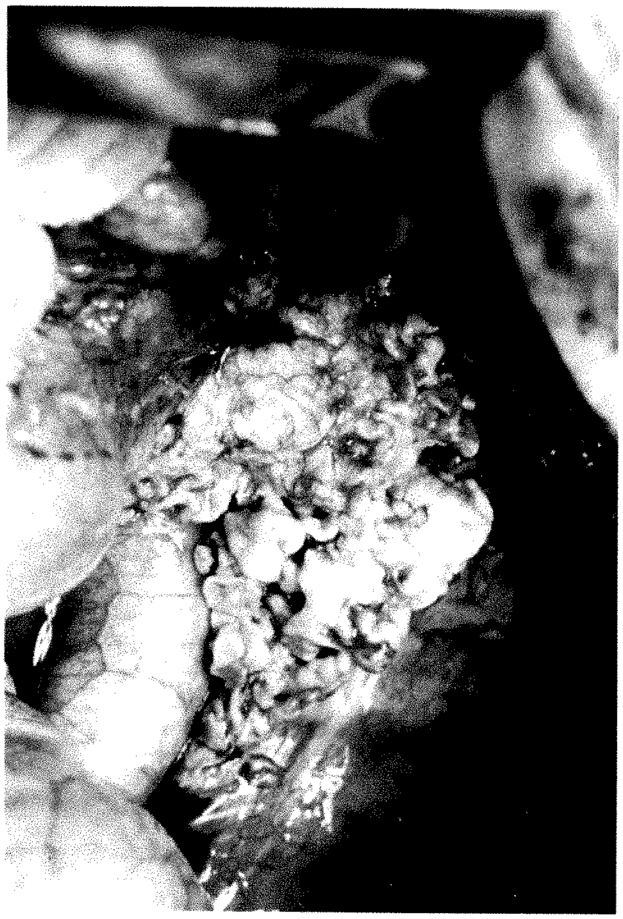

Fig 2. Intraoperative appearance of largest chondrosarcoma (8 × 5 × 5 cm) arising from parietal pleura cephalad and adjacent to proximal descending thoracic aorta.

within a year of diagnosis. Temporary regressions using doxorubicin and cyclophosphamide have been shown in these chondrosarcomas, as well as in those arising from the soft tissues of the extremities [4]. Although the prognosis of chondrosarcomas can be related to the degree of differentiation, even low-grade tumors are capable of recurrence, occasionally as late as 10 to 12 years after diagnosis.

Isolated cases of chondrosarcoma have been reported in the heart [5, 6] and in the fibrous capsule surrounding Lucite balls placed 20 years previously for plombage [7]. Pleural seeding is also extremely rare but has been reported in an osseous chondrosarcoma arising from the rib [8].

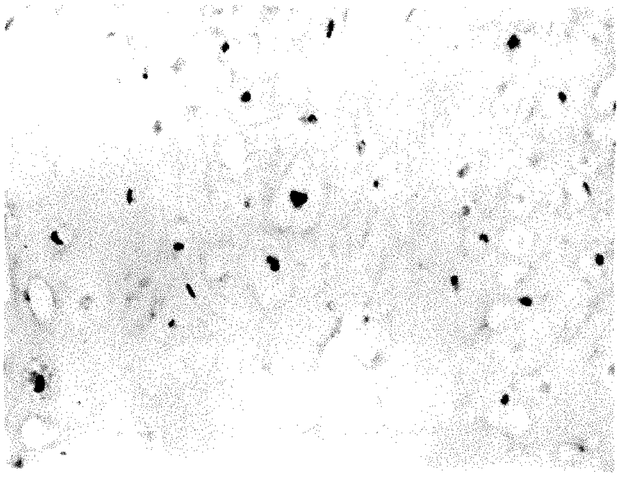

Fig 3. Histology of excised chondrosarcoma: well-differentiated chondrocytes in lacunae with prominent atypical nuclei, some binucleated. (Hematoxylin and eosin; ×450 before 48% reduction.)

Although no generalizations on tumor behavior can be based on an isolated case, the clinical findings, 13-year recurrence-free survival, and a comparison with similar low-grade chondrosarcomas after resection suggest a relatively benign potential for pleural chondrosarcoma. Wide local excision of this chondrosarcoma and all pleural implants therefore appears to be justified.

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Uncommon Intrathoracic Extrapulmonary Tumor: Primary Hemangiopericytoma

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We report an unusual case of primary intrathoracic extrapulmonary hemangiopericytoma. Despite the large size and rapid growth of the tumor, no histological sign of malignancy was present. Tumor cells immunostained positively only to vimentin.

(Ann Thorac Surg 1990;49:998–9)

Among the soft tissue tumors, hemangiopericytoma is quite rare [1, 2]. Thoracic localization is uncommon: a limited number of primary pulmonary hemangiopericytomas are reported in the literature [3–5] and the reports of intrathoracic extrapulmonary cases are even more rare [6]. The intrathoracic extrapulmonary primary hemangiopericytoma illustrated here is characterized by unusual size and rapid growth.

A 50-year-old woman had a dry cough for about 4 months before admission to the hospital. Chest roentgenogram (Fig 1) and computed tomographic scan showed a large, solid mass occupying about two thirds of the right pleural cavity. Fibrobronchoscopy showed extrinsic compression of the main and intermediate right bronchus. Perfusion scanning showed a defect of the lower, middle, and part of the upper right lobe. No other localization was detected.

At operation, the lesion was a mass of 35 × 25 cm (Fig 2) with a vascularized peduncle originating from the intercostal vessels without pulmonary and pleural adhesions. The lung reexpanded rapidly and completely after the removal of the tumor. There were no postoperative complications such as prolonged postoperative chest tube drainage or chylothorax.

Grossly, the tumor was a reddish, well-encapsuled mass weighing 1,350 g. When cut, the mass was found to be compact with very few and small cystic spaces and with no necrosis or hemorrhage. Histological sections were stained with hematoxylin and eosin and with Gomori. At the immunohistochemical level, the following antigens were investigated: cytokeratins, vimentin, S-100 protein, factor VIII-related antigen, actin, and α_1-antichymotrypsin.

On histological examination, the tumor was made up of a densely compact cellular proliferation with numerous small, medium, and large ramified vascular channels lined with flat endothelial cells. The neoplastic cells, uniform in size, showed round to ovoid nuclei and indistinct cytoplasmic borders (Fig 3). No pleomorphism or atypia was detectable. Degenerative stromal changes occurred but neither necrosis nor hemorrhage was present. Gomori staining showed a dense meshwork of reticulin fibers, thus confirming the perivascular distribution of tumor cells. The mitotic count was very low. Tumor cells immunostained positively only to vimentin.

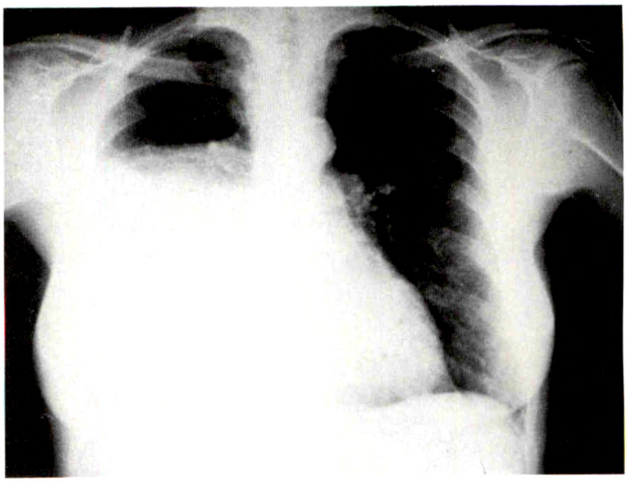

Fig 1. Chest roentgenogram showing the tumor mass largely occupying the right hemithorax.

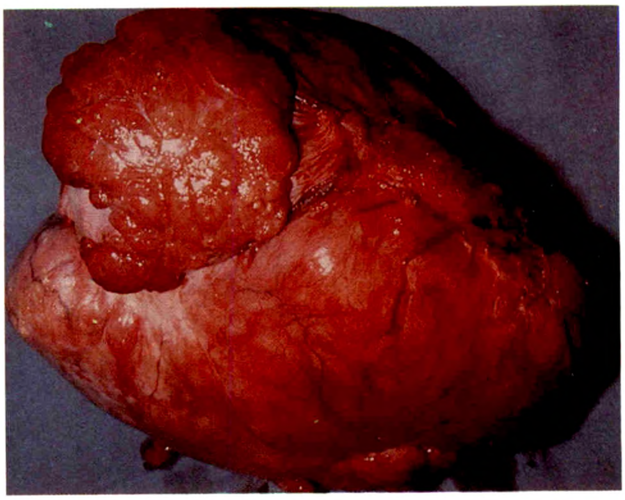

Fig 2. Gross view of the tumor.

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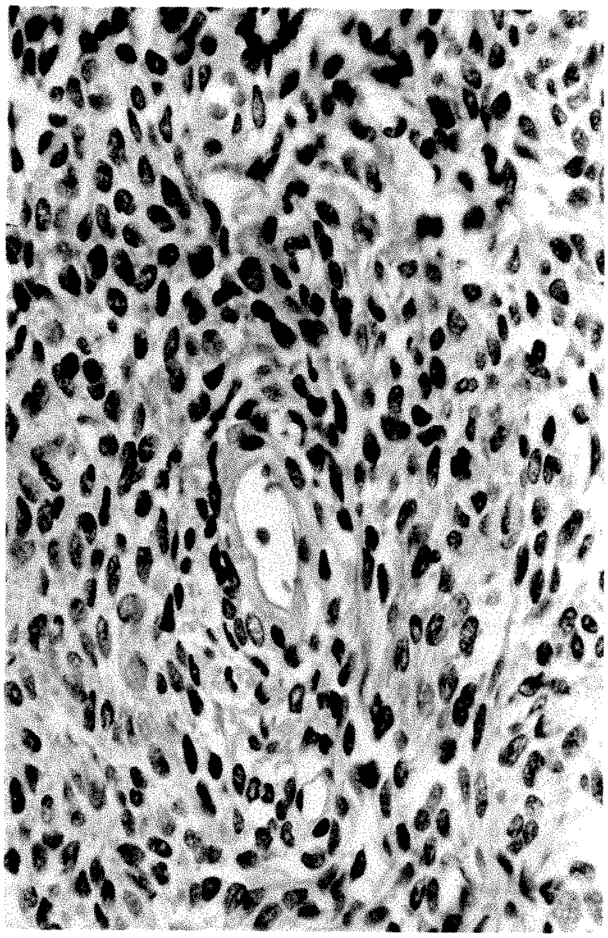

Fig 3. Neoplasm consists of a compact proliferation of pericytes having uniform ovoid to round nuclei and presenting no atypia or mitoses. (Hematoxylin and eosin, ×150).

A follow-up of perfusional scanning was performed 30 days after operation showing a healthy right lung. Six months after operation, the patient was well with no sign of recurrence.

Comment

This case represents a primary intrathoracic extrapulmonary hemangiopericytoma. Besides the rare localization, this tumor had an unusual size. The clinical findings (the onset of symptoms lasted only a few months; the lung fully reexpanded and was found healthy at postoperative perfusional scanning) excluded the idea of a longstanding compression of the tumor on the lung. Thus, we may reason that the neoplasm grew rapidly. However, there were no histological signs showing a high rate of proliferation (numerous mitoses, necrosis, and hemorrhage). This confirms the unreliability of histological features in correlation with the biological behavior of this type of neoplasia as the prognosis is related to tumor size, localization, and resectability [7]. The immunohistochemical findings (positive immunostaining only to vimentin) confirm the pattern reported in the literature [8].

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Right Atrial Myxoma Originating From the Inferior Vena Cava

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A patient undergoing successful excision of a right atrial myxoma arising from the inferior vena cava is reported. The rarity of this case prompted a review of the literature in which only 2 other patients with a right atrial myxoma originating from inferior vena cava tissue were found.

(Ann Thorac Surg 1990;49:1000–2)

Myxomas are the most frequent benign intracardiac tumors, most commonly arising from the left atrium [1]. Right atrial myxomas are much less common and are usually attached to the interatrial septum [2]. We report here a patient whose unusual feature was the presence of a right atrial myxoma originating from the inferior vena cava (IVC).

A 33-year-old woman was admitted to another hospital in November 1988. Approximately 2 months previously she had delivered at term a healthy baby without complications. During the last trimester of gestation, the patient started complaining of fatigue and mild exertional dyspnea; at that time ankle swelling and increased blood pressure were first noted. After delivery, she was observed to have persistent peripheral edema and hypertension suggesting further evaluation. On admission, physical examination showed a blood pressure of 165/115 mm Hg, a pulse rate of 104 beats per minute, and a 2/6 systolic murmur at the lower sternal border; the liver was palpable 4 cm below the right costal margin, and jugular venous distention was evident. The chest roentgenogram showed moderate enlargement of the cardiac shadow, and the electrocardiogram showed sinus tachycardia without major abnormalities. A standard two-dimensional echocardiogram showed the presence of a large mass almost filling the right atrium, extending to the IVC and entering into the right ventricle during diastole (Fig 1); however, the precise site of tumor insertion could not be identified.

The patient was transferred to our unit where urgent operation was performed on November 5, 1988. Cardiopulmonary bypass was instituted by individual cannulation of both venae cavae through the right atrium, avoiding any manipulation of the heart. With the patient under moderate systemic hypothermia (28°C) and after cold cardioplegic arrest of the heart, the right atrium was opened, and a large friable mass was noted to originate with a short stalk from inside the suprahepatic segment of the IVC. It became apparent that to get better exposure of the tumor insertion the IVC cannula should be temporarily removed; this was achieved after the nasopharyngeal temperature had been lowered to 25°C. During a brief period of low-flow perfusion, the mass was excised together with the entire pedicle and the underlying endothelium; the low attachment of the tumor prevented, however, a full-thickness excision of the IVC. The IVC cannula was then reinserted, the patient rewarmed, and the operation completed uneventfully.

Pathologic examination showed a large, friable multilobulated mass, which at histological examination showed the classic features of a myxoma (Fig 2).

Comment

Right atrial myxomas usually originate from the fossa ovalis or at the base of the interatrial septum. In such situations, surgical exposure of the tumor is usually easier and its excision simpler when compared with that of a left atrial myxoma [1]. The patient described herein had the unusual finding of a right atrial myxoma arising from the suprahepatic segment of the IVC; this occurrence is quite exceptional and was not mentioned in recent reviews of patients with intracardiac myxomas [1, 3]. Right atrial myxomas originating from IVC tissue have been previously reported only twice (Table 1). Devig and colleagues in 1980 [4] described a 28-year-old man who underwent excision of a right atrial myxoma inserted at the junction between the right atrial wall and the anterosuperior aspect of the IVC. The tumor was removed together with a segment of IVC. More recently, Cujec and associates [5] reported a 25-year-old man with a right atrial myxoma originating from the eustachian valve and prolapsed into the IVC causing Budd-Chiari syndrome. The mass was successfully excised, but details of the surgical technique were not mentioned.

The peculiar location of a right atrial mass, as in the present case, may pose specific surgical problems. One of these concerns the best technique of venous cannulation to prevent fragmentation and possible pulmonary embolization. To minimize this potential complication, venous return can be accomplished by cannulating the superior

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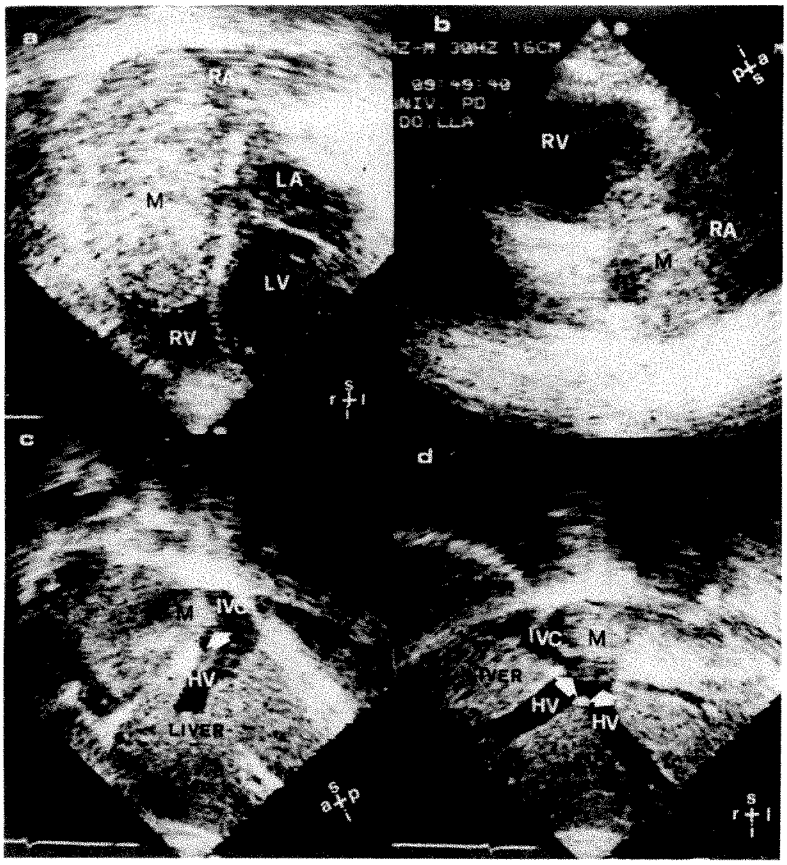

Fig 1. Two-dimensional echocardiogram showing a large mass (M) *filling the right atrium* (RA) *(a, b) and protruding into the inferior vena cava* (IVC) *(c, d).* (HV = *hepatic vein;* LA = *left atrium;* LV = *left ventricle;* RV = *right ventricle.)*

vena cava and a femoral vein [4]. In the present case, we used direct caval cannulation, as previously done in all other patients with a right atrial myxoma [6], because the exact site of tumor insertion was not identified on preoperative echocardiogram. We have found that this technique can be used without complications even in the presence of large intraatrial masses, provided that undue manipulation of the heart is avoided. When the myxoma is inserted deep into the IVC, as in the present case, temporary removal of the IVC cannula is needed to obtain a better exposure of the mass. However, had the precise site of tumor attachment been identified using intraoperative transesophageal echocardiography, which was not available for our patient, a different approach by cannulation of a femoral vein would have been probably more appropriate.

To avoid recurrences, a radical operation includes removal of the myxoma and its entire base of implantation, which is easily performed when the tumor is inserted into the fossa ovalis or the free atrial wall. In the present case, however, full-thickness excision was prevented by the unusual attachment of the mass into the IVC. Despite the absence of clear signs of local infiltration, periodic noninvasive monitoring of this patient seems mandatory.

Table 1. Summary of Patients With a Right Atrial Myxoma Arising From Inferior Vena Cava Tissue

Author	Age (yr)	Sex	Symptoms	Diagnosis	Location	Surgical Technique
Devig et al (1980) [4]	28	M	Exertional dyspnea	Cardiac catheter	IVC–RA junction	Full thickness excision
Cujec et al (1987) [5]	25	M	Budd-Chiari syndrome	Intraoperative	Eustachian valve	Not mentioned
Bortolotti et al (present case)	33	F	Exertional dyspnea, peripheral edema	2-D echocardiography	Suprahepatic IVC	Excision

IVC = inferior vena cava; RA = right atrium; 2-D = two-dimensional.

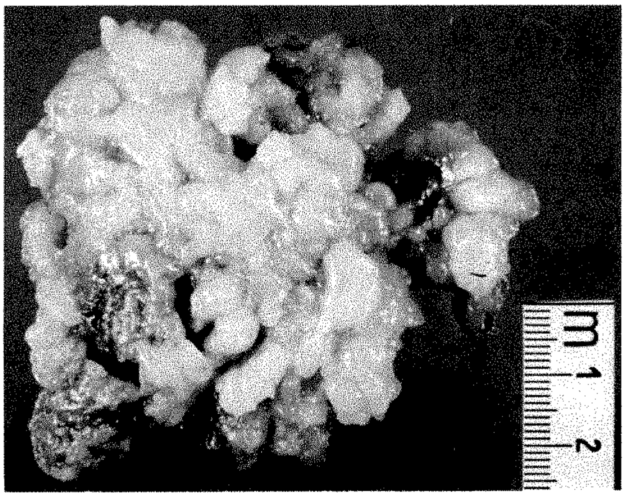

Fig 2. Gross appearance of the excised myxoma.

We are indebted to Richard Van Praagh, MD, for help during the preparation of the manuscript and to Gian Carlo Pengo for technical assistance.

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Partial Biventricular Repair for Double-Outlet Right Ventricle With Left Ventricular Hypoplasia

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The presence of left ventricular hypoplasia in double-outlet right ventricle may increase the risk of biventricular repair and Fontan procedures. The hypoplastic left ventricle of an 11-year-old girl with complex double-outlet right ventricle was successfully incorporated in a modified biventricular repair by Dacron patch closure of the ventricular septal defect, diversion of venous return of the inferior vena cava to the mitral valve with an intraatrial baffle, insertion of a left ventricular apex to pulmonary artery valved aortic homograft, and diversion of residual systemic venous return directly to the lungs with a bidirectional superior vena cava to pulmonary artery shunt. Postoperatively, the systemic venous atrial pressure was 6 mm Hg and the pulmonary artery pressure was 14 mm Hg. This method is proposed as a partial biventricular repair hemodynamically superior to the Fontan procedure, although aortic homograft revision may be required in the future.

(Ann Thorac Surg 1990;49:1003–5)

Most forms of complex double-outlet right ventricle (DORV) are now amenable to biventricular repair with insertion of an intraventricular baffle or arterial switch procedure [1]. Double-outlet right ventricle with left ventricular (LV) hypoplasia has been treated with a modified Fontan procedure [2]. We present an alternative method of biventricular repair using a hypoplastic left ventricle as the pulmonary ventricle by diverting systemic venous return of the inferior vena cava (IVC) to the mitral valve (MV) and reducing left ventricular volume load with a bidirectional superior vena cava (SVC) to pulmonary artery (PA) shunt.

An 11-year-old girl with DORV, a noncommitted ventricular septal defect (VSD), d-transposition of the great arteries, pulmonary atresia, intact atrial septum, and parachute MV was referred for definitive surgical repair. A central aorta to PA shunt had been placed and revised when the patient was a newborn. Classic left and right Blalock-Taussig shunts were performed when the patient was 3 and 5 years of age, respectively. She was cyanotic with a hemoglobin level of 174 g/L and had moderate exercise intolerance, yet she was on no medication. The chest roentgenogram showed several thoracic hemivertebrae and mild cardiomegaly with a minimal degree of symmetric pulmonary venous congestion. An electrocardiogram demonstrated sinus rhythm with a superior counterclockwise frontal axis and right ventricular hypertrophy. Remarkable echocardiographic findings were an intact atrial septum, an apex-forming left ventricle, and a parachute abnormality of the MV with an annulus of 15 mm and a diastolic velocity of 1.8 m/s. Heart catheterization data included an SVC and right atrial saturation of 67%, an aortic saturation of 79%, and pressures in the right ventricle of 120/5 mm Hg and PA of 22/14 mm Hg (mean 16 mm Hg). There was no angiographic evidence of a left SVC, important systemic to PA collaterals, or tricuspid valve regurgitation. Ventricular volumes were not measured; however, the left ventricle was approximately half the size of the right ventricle on levophase. The decision for biventricular repair or for a modified Fontan was dependent on intraoperative assessment of the left atrial pressure, MV morphology, LV size, and VSD position.

Intraoperatively, mean pressures measured in the PA, right atrium, and left atrium were 18, 4, and 14 mm Hg, respectively. There was a parachute MV with an annulus of 17 mm. The VSD was noncommitted and the left ventricle appeared mildly hypoplastic with a relatively thin posterior wall. In view of this, the left ventricle was not used as a systemic ventricle and a modified biventricular repair was performed in the following stages: (1) division of the central shunt and closure of the Blalock-Taussig shunts with large hemoclips, (2) Dacron patch closure of the VSD through the right atrium and tricuspid valve, (3) tricuspid valve annuloplasty, (4) atrial septectomy, incision of the coronary sinus into the left atrium, and placement of a Gore-Tex baffle (W.L. Gore and Associates, Inc, Flagstaff, AZ) from the MV along the posterior wall of the left atrium and right atrium to the IVC (Fig 1A), (5) insertion of an LV apex to PA conduit using a 20-mm albumin-impregnated Dacron graft (Bard albumin-coated, Debakey Vasculour II straight vascular prosthesis; Bard Cardiosurgery Division, Billerica, MA) attached to an 18-mm valved aortic homograft, and (6) a bidirectional SVC to PA shunt (Fig 1B). The proximal conduit was sutured to an apical incision between the left

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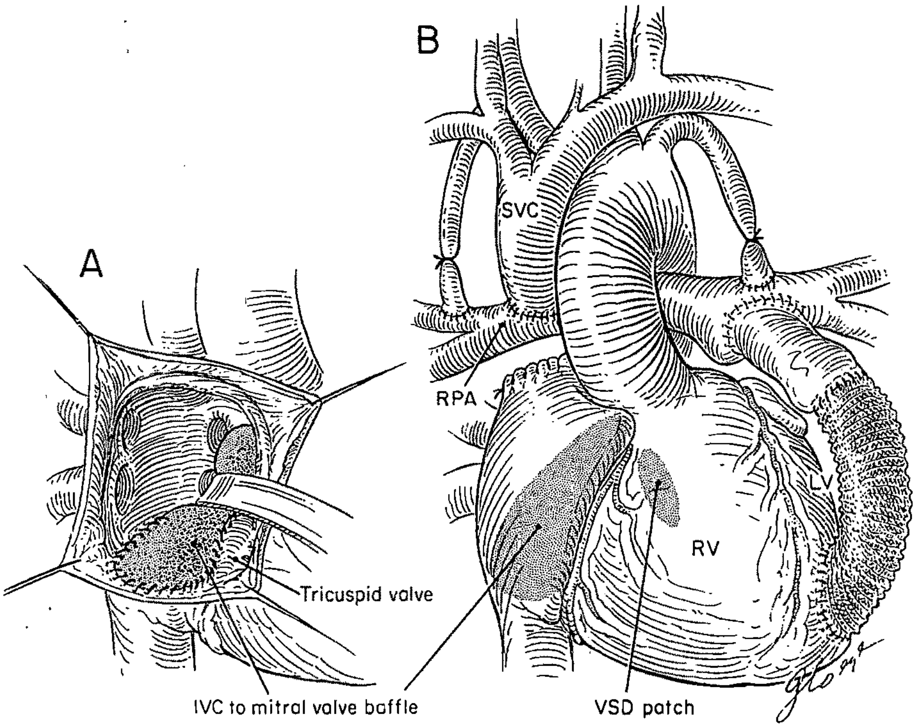
Fig 1. Partial biventricular repair of double-outlet right ventricle: (A) intraatrial baffle diverting systemic venous return from the inferior vena cava (IVC) to the mitral valve and pulmonary venous return to the tricuspid valve and right ventricle (RV); (B) patch closure of the ventricular septal defect (VSD), Dacron graft/valved aortic homograft inserted between the left ventricular (LV) apex and the pulmonary artery, and bidirectional superior vena cava (SVC) to right pulmonary artery (RPA) anastomosis.



anterior descending and diagonal coronary arteries and distally anastomosed with the main PA. There were no intraoperative complications. Cardiopulmonary bypass was 156 minutes with an aortic cross-clamp time of 125 minutes. Intermittent cold-blood cardioplegia was used with warm-blood cardioplegia reperfusion.

The patient came off bypass in sinus rhythm with SVC and PA pressures of 14 mm Hg and an IVC pressure of 6 mm Hg. She was managed with low doses of inotropic support, diuretic agents, and afterload reduction. Antiplatelet therapy with aspirin and dipyridamole was started on the third postoperative day. She was discharged on the eighth postoperative day. Three months after operation, she had no hepatomegaly, and echocardiography showed good ventricular function and no obstruction of the IVC to MV baffle.

Comment

Double-outlet right ventricle is occasionally associated with LV hypoplasia and not amenable to repair with an intraventricular tunnel, an atrial switch, or an arterial switch procedure. Russo and colleagues [2] reported improved results in managing complex forms of DORV with a modified Fontan procedure; however, LV hypoplasia remained a negative prognostic feature. Systemic venous hypertension of varying degrees is a disadvantage of the Fontan operation and can be associated with low cardiac output and increased mortality in the early postoperative period [3]. An abnormal response to exercise may be evident postoperatively, and exposure of the right atrial wall to high pressure may eventually result in supraventricular arrhythmias [4]. A biventricular repair is therefore preferred whenever possible.

We propose a method in which a hypoplastic left ventricle can be used in the repair of complex forms of DORV and d-transposition of the great arteries with a VSD. This is done by reducing the volume load of the pulmonary ventricle with a bidirectional SVC to PA shunt. The VSD is closed and residual systemic venous return is diverted to the hypoplastic left ventricle by performing an intraatrial baffle from the IVC to the MV. The LV apex and PA are connected with a valved homograft when pulmonary atresia or LV outflow obstruction is present. A valved homograft may require revision if late valve deterioration occurs. However, valvar regurgitation of a low-pressure pulmonary system may be tolerated and balloon valvoplasty may be considered if the valve or conduit becomes obstructive.

The advantage of this procedure is that right atrial and IVC pressures are reduced when venous return flows in series with a functional pulmonary ventricle. The potential for supraventricular arrhythmias may be decreased and the response to exercise may be improved. The concept of incorporating a hypoplastic ventricle as part of a biventricular repair has also been applied to patients with right ventricular hypoplasia associated with pulmonary atresia and intact ventricular septum [5]. A bidirectional SVC to PA shunt has been useful with this procedure to reduce the volume load of the hypoplastic right ventricle [5]. The degree of MV and LV hypoplasia contraindicating a complete biventricular repair yet allowing a "partial" biventricular procedure is not known. Our experience with the hypoplastic right ventricle suggests that individuals with MV and LV sizes approximately one half to two thirds of normal may benefit from this procedure, provided the pulmonary vascular resistance is not excessive. Quantitative assessment of LV size, compliance, wall thickness, and function in individuals with DORV or transposition of the great arteries and VSD may determine the degree of LV hypoplasia suitable for a partial biventricular repair.

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Melanotic Schwannoma of the Sympathetic Ganglia: Pathological and Clinical Characteristics

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Reports of melanin-producing tumors of Schwann cell origin are extremely rare. Reports of only 9 such tumors arising from sympathetic ganglia have been published previously. Two new cases of melanotic schwannoma of sympathetic ganglia are reported. The pathological and clinical characteristics of these unusual melanotic tumors are discussed.

(Ann Thorac Surg 1990;49:1006–8)

Neurogenic tumors, occurring in association with spinal nerves or sympathetic ganglia, account for 15% to 30% of primary mediastinal tumors and 5% of retroperitoneal tumors. The majority of these tumors arise in the vertebral foramen or paravertebral sulcus. Included among neurogenic tumors are benign entities such as neurofibroma, ganglioneuroma, and schwannoma, and malignant lesions such as neuroblastoma and neurosarcoma (malignant schwannoma). Rarely, schwannomas may produce melanin, giving them a dark brown or black appearance. Only 9 patients with melanotic schwannoma arising from sympathetic ganglia have been recorded, and thus, the biological behavior of the autonomic lesions is not completely understood. This report describes 2 additional patients with melanotic schwannoma of the sympathetic ganglia and reviews the pathological and clinical characteristics of these unusual tumors.

Patient 1

An asymptomatic 20-year-old male college athlete was found to have a right posterior mediastinal mass on chest roentgenography in 1987. Preoperative magnetic resonance imaging demonstrated the mass with no invasion into the neural canal (Fig 1). A 4-cm spherical mass was resected along with a portion of the sympathetic chain through a right posterolateral thoracotomy. No gross invasion of the vertebral body or of the intervertebral disc space was noted.

Examination of the resected specimen showed a 4 × 2.8 × 2.5-cm, tan, encapsulated mass that had a black, focally hemorrhagic smooth surface when cut. Microscopically, tumor cells were seen penetrating the fibrovascular capsule but not extending through it. Spindle-shaped tumor cells and small tumor cells resembling lymphocytes predominated. Also present were cells with large basophilic nuclei, prominent nucleoli, and abundant cytoplasm resembling ganglia cells. Mitoses were rarely seen. Several cells were multinucleated and had large bizarre nuclei. Melanin pigment was identified in varying amounts in many cells. Numerous macrophages contained melanin and hemosiderin. Stains for S-100 (immunoperoxidase) before and after bleaching of melanin pigment showed areas of positivity among the tumor cells.

The patient's postoperative course was entirely unremarkable except for a right Horner's syndrome. Follow-up chest roentgenography at 6, 12, 18, and 24 months, and magnetic resonance imaging at 24 months have indicated no evidence of recurrence.

Patient 2

A 36-year-old woman was seen in February 1988 with an acute episode of right infrascapular pain radiating to her right subcostal region. Physical examination showed a soft abdomen with mild voluntary guarding and tenderness in the right upper quadrant. There was no discernible mass. She had no lower-extremity edema or tenderness, and neurological examination was normal.

Abdominal ultrasound showed a large mass behind the liver. Computed tomography confirmed this finding and demonstrated a 10-cm mass in the right paravertebral area, displacing the right kidney inferiorly and the right lobe of the liver and inferior vena cava anteriorly and laterally (Fig 2). Urine levels of catecholamines, metanephrines, and vanillylmandelic acid were within normal limits. Angiography demonstrated close adherence of the tumor to the right kidney and confirmed anterolateral displacement of the inferior vena cava. There were two functioning kidneys on pyelography.

Resection of the tumor was accomplished through a right thoracoabdominal approach. Grossly, the tumor invaded the prevertebral fascia, and the right renal artery was encased by tumor for a 1-cm segment, requiring partial resection with reanastomosis. The right adrenal gland was adherent to but not invaded by the tumor.

Tissue examination showed a 7 × 11 × 7.5-cm encapsulated mass that appeared gray and lobulated with areas of hemorrhage and necrosis on cut section. Microscopic examination showed elongated spindle-shaped cells with poorly defined cytoplasmic borders arranged in interlacing fascicles. The nuclei were elongated with blunted ends and mitotic figures were present. Pigmentation

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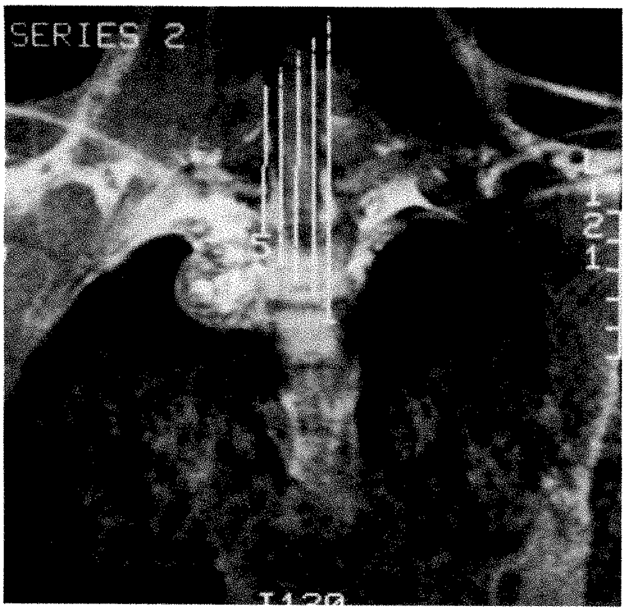

Fig 1. Magnetic resonance image of melanotic schwannoma arising from sympathetic ganglia high in the right posterior mediastinum.

varied from a fine granular dusting to heavy clumps that obscured the nucleus. Prominent vasculature was noted, and the vessel walls were invaded by nests of malignant cells. Immunochemical stains showed nuclear and cytoplasmic staining for S-100, and cytokeratin was not identified.

On electron microscopy, premelanosomes and melanosomes were scattered throughout the cytoplasm and were numerous in some cells. Occasional junctional structures and focally prominent basal lamina were present. Neurosecretory granules and cytoplasmic filaments were not seen.

The patient did well and was discharged on the 10th postoperative day. A follow-up at 18 months showed evidence of recurrent tumor adjacent to the right kidney and inferior vena cava. The patient is currently receiving chemotherapy.

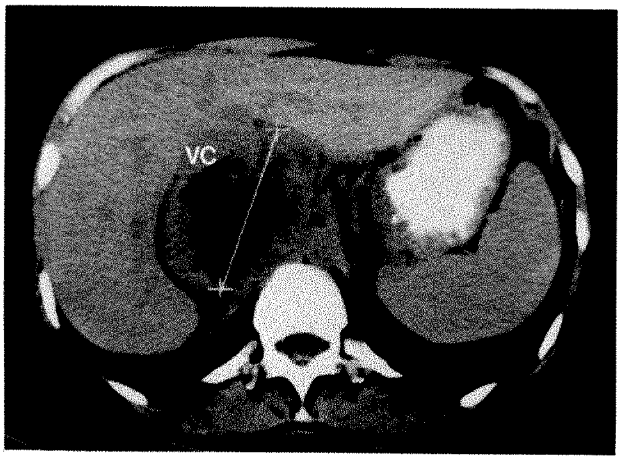

Fig 2. Computed tomographic scan of the abdomen demonstrating melanotic schwannoma in the retroperitoneum. There is anterior and lateral displacement of the vena cava (VC).

Comment

Primary melanotic tumors of sympathetic ganglia are extremely rare. In 1932, Millar [1] reported the first case. Since that time, reports of 8 additional cases have been published [2–6]. Tumors arise along the sympathetic chain, most commonly in the thorax, although cervical and lumbar tumors have also been reported. The tumors vary in size and invasiveness but all have a characteristic dark brown or black appearance. Histological features include hypercellularity, cellular pleomorphism, and spindle cells arranged in fascicles. Melanin pigmentation, which gives the tumor its color, varies from area to area but can be dense enough to obscure all cellular detail. Immunohistochemical and ultrastructural studies identify these tumors as Schwann cell in origin. The tumor stains with both S-100 and neuron-specific enolase [6]. Ultrastructural features include melanosomes in membranes and variable basal lamina production. The degree of differentiation is reflected in the amount and continuity of basement-membrane production [4]. In some instances, mitotic activity, necrosis, and invasive characteristics are noted and correlate with clinical aggressiveness [3–5].

Clinical presentation is related to tumor size, proximity to adjacent structures, and presence of hematogenous metastasis. Computed tomography and magnetic resonance imaging are useful in evaluating tumor burden. Radionucleotide bone scanning may be used to identify bone metastasis. Optimal treatment of pigmented schwannomas involving the sympathetic ganglia has not been defined but complete excision of all tumor is presently indicated. Adjuvant treatment modalities including chemotherapy and irradiation are not well established [3, 6].

One of our patients has had local recurrence of her tumor. This is typical of the natural history of melanotic schwannomas involving sympathetic ganglia. The patient reported by Millar had paresthesias due to spinal cord invasion and died of extensive metastases to lung, liver, bones, and adrenal glands [1]. One of the patients reported by Fu and associates [3] had loss of sympathetic nervous function in one leg, underwent removal of a mass extending from L-3 to L-5, and died of extensive intrathoracic metastatic disease. Six of 9 previously reported patients died of complications of their disease, generally related to widely metastatic tumor. Patterns of metastases would indicate that tumor spread is by direct invasion or hematologic spread with involvement of lung and bone [1, 3, 6].

Melanotic schwannomas have also been described arising from spinal and peripheral nerves. The tumors arising from these structures tended to be symptomatic early and were detected at a relatively smaller size, relating to nerve dysfunction or mass effect. Cure by local excision has been reported in patients with peripheral pigmented schwannomas [7, 8]. Whether there is a biological difference between pigmented tumors of Schwann cell origin arising from the autonomic system and those arising from sensory/motor nerves is unresolved. Melanotic schwannomas arising from sympathetic ganglia do appear to be more aggressive clinically than those arising peripherally.

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Multiple Aneurysms of the Coronary Arteries in a Patient With Systemic Aneurysmal Disease

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A case of multiple coronary artery aneurysms in a patient with an abdominal aortic aneurysm and bilateral popliteal artery aneurysms is reported. As diagnosis and treatment of coronary artery disease become more aggressive, a higher incidence of detection of coronary artery aneurysms in conjunction with peripheral vascular lesions is likely. The role of coronary sinus cardioplegia in minimizing the risk of embolization from the coronary aneurysms during surgical repair is emphasized.

(Ann Thorac Surg 1990;49:1009–10)

Since the first report of coronary artery aneurysms, several investigators have presented thorough reviews of the literature regarding this relatively rare vascular defect [1, 2]. In 1971, Ebert and associates [3] performed the first successful surgical repair of coronary artery aneurysms in a 31-year-old woman. This paper reports a case of multiple coronary aneurysms as a manifestation of diffuse aneurysmal disease in an elderly man.

A 75-year-old man was initially referred to the New York University Medical Center in October 1988 for evaluation of an enlarging abdominal aortic aneurysm. Abdominal ultrasound and computed tomographic scans revealed a 6-cm infrarenal aneurysm, and on routine preoperative workup, the patient was noted to have an ejection fraction of 0.30. A cardiac catheterization was performed, which showed aneurysmal dilatations of the proximal coronary arteries and distal occlusive disease. The left main coronary artery was dilated in its midportion to approximately 3.5 cm, and the left anterior descending artery was totally occluded. Also, the proximal branches of the left circumflex artery were severely obstructed (Fig 1). A second coronary aneurysm, 7 cm in diameter, was found in the proximal right coronary artery approximately 2 cm from a severely stenotic coronary ostium (Fig 2). Ultrasonography of the lower extremities showed bilateral popliteal aneurysms approximately 2 cm in diameter and severely ectatic femoral arteries without focal dilatation. Examination of the patient was remarkable for a 6 to 7 cm pulsatile, supraumbilical mass.

At operation, a large (7 to 8 cm) right coronary artery aneurysm extending to the bifurcation and a smaller (3 to 4 cm) left main coronary artery aneurysm were noted (Fig 3). To minimize the possibility of distal embolization from either aneurysm, cold-blood cardioplegia was injected through the coronary sinus under low pressure, and iced saline solution was applied topically to the heart. A prompt diastolic arrest was obtained with a myocardial temperature range of 7° to 11°C. Saphenous vein bypass grafting was performed with the distal anastomoses to the circumflex marginal and anterior descending arteries. Cold blood was injected down each graft after the distal anastomosis, and then the proximal anastomoses were constructed. After the proximal veins were attached to the aorta, the aneurysms were evaluated. Because the aneurysm in the left main coronary was circumferentially calcified and rigid to palpation and was the source of large septal and important circumflex marginal branches, it was left undisturbed. The right coronary artery aneurysm was opened, revealing a laminated thrombus contained within an exceedingly thin and partially necrotic adventitial wall. After the thrombus and debris were removed, the proximal and distal ends of the coronary artery were oversewn and the aneurysm wall was closed after its edges were excised. A vein graft was attached to the posterior descending artery. The aorta was then unclamped, and the patient was weaned from cardiopulmonary bypass with minimal inotropic support. The patient made a rapid and uneventful recovery and subsequently underwent a successful repair of his abdominal aortic aneurysm.

Comment

The incidence of coronary artery aneurysms has been reported as 1.4% based on postmortem studies [2]. Such aneurysms occur three times as frequently in male patients as in female patients, are noted with approximately equal frequency in the right and left coronary vessels, and are multiple in 25% of patients [2]. Daoud and co-workers [2] reported the association of abdominal aortic aneurysms with coronary artery aneurysms and noted that rupture of an abdominal aortic aneurysm was the cause of death in 8 of the 10 patients they reported.

Atherosclerosis plays an important role in the development of coronary artery aneurysms and may be a predominant cause in about half of all patients. The pathogenesis of coronary artery aneurysms is most likely related to injury of the intima overlying intrinsic disease of the media, causing dilatation of the vessel and intimal ulceration [4, 5]. Ventricular dysfunction results from distal embolization of microthrombi, resulting in progressive ischemic loss of myocardium [6].

No clinical picture is specifically suggestive of the

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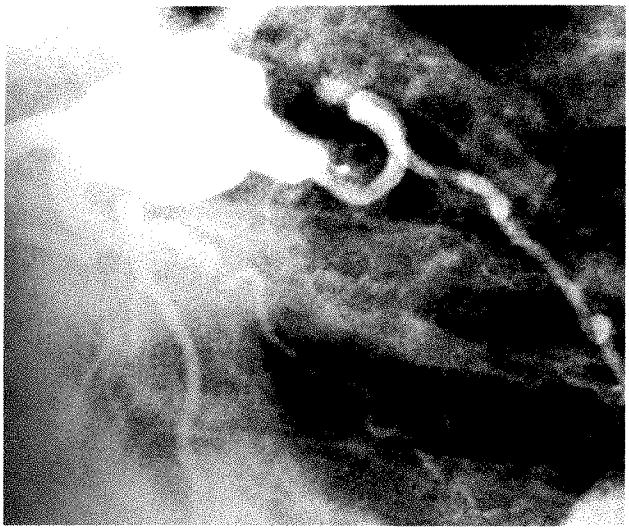

Fig 1. Angiogram of left coronary artery demonstrating aneurysm.

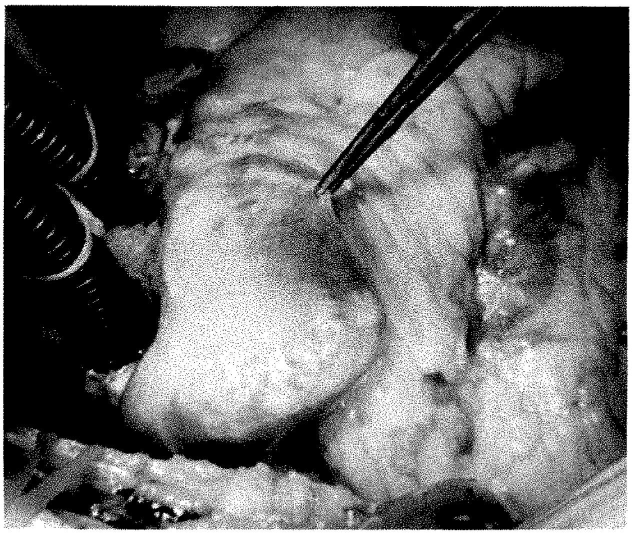

Fig 3. View at operation of large right coronary artery aneurysm.

diagnosis of coronary artery aneurysm. Many patients are first seen with angina or myocardial infarction due to underlying coronary obstruction or congestive heart failure as a result of progressive ventricular dysfunction. Currently, the diagnosis is most often an unexpected finding at cardiac catheterization. If left untreated, the prognosis for coronary artery aneurysms is poor [2]. Myocardial infarction and rupture of the aneurysm are both well documented outcomes [1, 3]. In their review of the Coronary Artery Surgery Study (CASS), Swaye and associates [7] noted that patients with aneurysmal disease had a significantly higher incidence of myocardial infarction than a control group with only coronary stenosis (53% versus 47%). Surgical correction is the only definitive treatment and is clearly indicated in most instances.

This report highlights two important points. First, aneurysmal disease of the arteries is truly a systemic disease. It is well known that when an aneurysm occurs in the peripheral vasculature, multiple aneurysms are the rule [8]. It is surprising that so few cases of coronary artery aneurysm have been reported in conjunction with diffuse aneurysmal disease, and it seems likely that many more cases would be detected if an active search for coronary aneurysms were made in every patient with multiple peripheral aneurysms. It can be expected that coronary artery aneurysms will be seen more frequently by cardiac surgeons in the future as the diagnosis and treatment of coronary artery disease becomes more aggressive. Second, it should be noted that retrograde coronary sinus cardioplegia can be used effectively and safely in such cases to provide adequate myocardial protection without risking distal embolization from the aneurysm during injection of cardioplegic solution. Coronary sinus cardioplegia should be strongly considered by the surgeon in any operative repair of a coronary aneurysm.

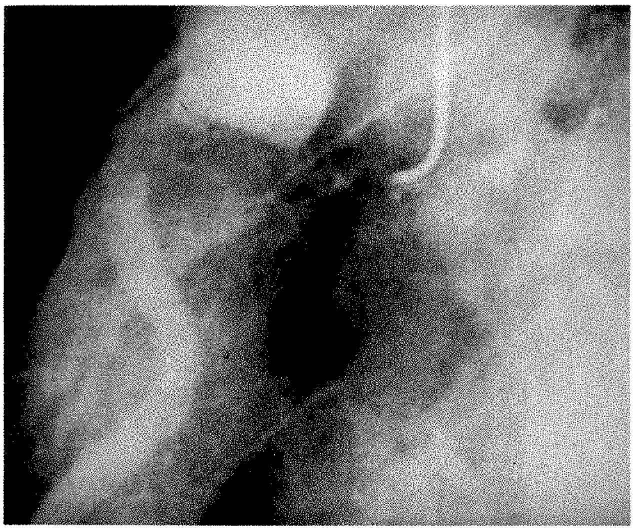

Fig 2. Angiogram of right coronary artery demonstrating diffuse aneurysmal dilatation. At operation the lumen contained a large laminated thrombus extending from the ostium to the bifurcation of the right coronary artery.

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Retrophrenic Location of the Internal Mammary Artery Graft

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A technique of locating the internal mammary artery behind the phrenic nerve is described, which extends the application of internal mammary artery grafting to the lateral wall of the left ventricle. The technique also facilitates the performance of the posterolateral anastomosis between the internal mammary and circumflex coronary arteries.

(Ann Thorac Surg 1990;49:1011–2)

The most direct route of the internal mammary artery (IMA) graft from its origin to the back of the heart is through the pericardium [1]. Using conventional surgical techniques, the IMA graft is passed around the free edge of the pericardium or through a V-shaped incision or pericardial window anterior to the phrenic nerve [2]. However, an alternative route to the posterior aspect of the heart is behind the phrenic nerve in front of the hilum of the lung. The retrophrenic technique is particularly valuable for grafting the circumflex marginal or intermediate coronary arteries and is occasionally useful for grafting the left anterior descending artery or the diagonal branch.

Technique

The IMA graft is mobilized with the pleura to the level of the subclavian vein or to a higher level if necessary. The endothoracic fascia is divided to free the subclavian vein anteriorly and laterally, to display the IMA passing superiorly behind the inferior border of the subclavian vein. The lateral costal branch of the IMA should be divided if present [3]. The IMA is divided after heparinization before cardiopulmonary bypass, and the flow is checked. The pericardium between the phrenic nerve and the anterior aspect of the hilum of the lung is exposed by sharp dissection of the overlying mediastinal pleura. A window is then created through a small recess in the pericardium between the phrenic nerve and the hilum of the lung in the region of the left atrial appendage using diathermy (Fig 1). Care is necessary to avoid damage to the left main pulmonary artery and superior pulmonary vein posteriorly or to the phrenic nerve anteriorly. This incision is lengthened to allow easy passage of the IMA pedicle, which enters the pericardium near the left atrial appendage (Fig 2). If necessary, the distal portion of the IMA is transected to avoid redundancy, leaving sufficient length of mammary artery for grafting to the appropriate coronary artery with the heart lifted out of the pericardium. This will usually result in an excess of IMA pedicle once the heart is returned to the pericardial sac. An anastomosis is then performed in the usual fashion, and the pedicle is anchored to the epicardium.

Comment

The retrophrenic technique provides a short route from the left IMA origin to the circumflex marginal or intermediate coronary artery and occasionally to the left anterior descending or diagonal coronary arteries when the heart is rotated to the left.

The advantage of the retrophrenic technique is that it is a direct route between the IMA origin and the terminal branches of the left coronary artery. The distal circumflex marginal branches can be reached using this technique. By using the proximal portion of the IMA, the largest blood flow is obtained. In addition, with the heart elevated the graft enters the pericardium from the posterolateral aspect and angles forward, thus facilitating the anastomosis. At the end of the procedure, the pericardium can be closed almost completely over the heart and great vessels with the IMA graft lying laterally, thus protecting both the heart and the graft from injury during closure or reopening of the sternum.

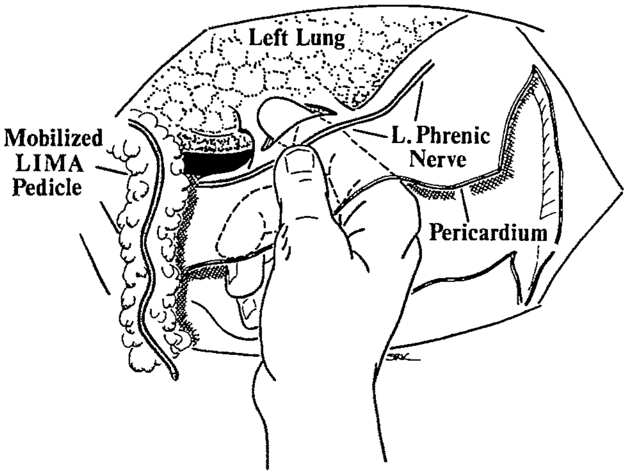

Fig 1. The tip of the right index finger is shown in the left pleural cavity anterior to the hilum of the lung immediately posterior to the phrenic nerve. (LIMA = *left internal mammary artery.)*

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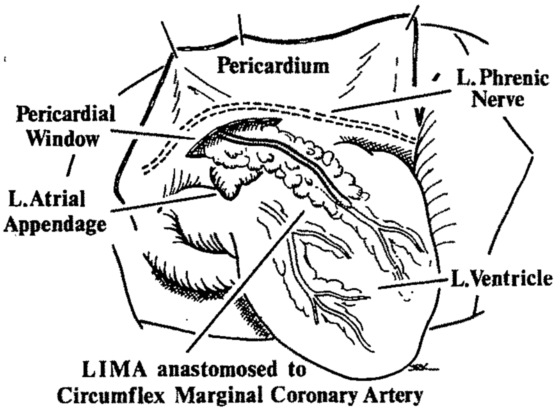

Fig 2. With the heart elevated, the left internal mammary artery (LIMA) *pedicle enters the pericardial cavity adjacent to the left atrial appendage and the LIMA is anastomosed to the circumflex marginal coronary artery.*

A disadvantage of this technique is that the phrenic nerve is more likely to be damaged, particularly if there is only a small space between the phrenic nerve and the anterior aspect of the hilum. Although elevation of the heart may result in temporary angulation of the IMA pedicle, this disappears when the heart is returned to the pericardium. At the end of the surgical procedure, hemostasis can be more difficult to achieve if bleeding occurs in the retrophrenic area.

If reoperation is required, it is usually not necessary to mobilize the retrophrenic IMA graft to provide a dry operative field. The IMA can be left untouched, and the operation can be performed with the patient under deep hypothermia. Adequate visualization of the coronary anastomoses can then be obtained by reducing the cardiopulmonary bypass flow without clamping the IMA pedicle.

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Technique for Visualization of the Functioning Mitral Valve

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A technique is described for visualization of the mitral valve in the beating perfused heart to identify areas of abnormality in the valve apparatus before reconstruction. Risks of air embolus and myocardial ischemia are minimized.

(Ann Thorac Surg 1990;49:1013)

Since Carpentier's description of techniques for reconstruction of the mitral valve, reconstruction has become the procedure of choice whenever it can be technically accomplished [1]. A problem in reconstruction, particularly for the surgeon just beginning to use reconstructive techniques, is identification and location of the specific valve pathology: (1) annular enlargement, (2) leaflet redundancy, (3) leaflet prolapse due to chordal lengthening, or (4) ruptured chordae. This can be difficult in the warm, fibrillating heart or in the cold, flaccid heart after the delivery of cardioplegic solution. A technique is described to allow visualization of the functioning mitral valve in the beating, perfused heart, which permits identification and location of the specific valve pathology without the risk of air embolization or myocardial ischemia.

After the institution of cardiopulmonary bypass, with cannulation of the ascending aorta and bicaval venous cannulation, a Y cannula (No. 20012 aortic root cannula, DLP, Inc, Grand Rapids, MI) for cardioplegia infusion is placed in the ascending aorta and a 3/8-inch (9.4 mm) polyvinyl tubing bridge is connected from a stopcock in the arterial line to the Y cannula (Fig 1). The aortic cross-clamp is placed, and the bridge is opened to allow perfusion of the aortic root. The left atrium is opened, and the atriotomy is extended to allow visualization of the mitral valve with aspiration of blood from the atrium. Blood from the beating ventricle is ejected into the atrium identifying the area of leakage. The valve apparatus can be inspected, and all pathological conditions can be easily and fully identified, generally in only two to three minutes. When the inspection process is complete, the bridge can be closed and the cardioplegic solution is infused for myocardial protection, and the operation is completed. This technique also allows identification of mitral valve pathology not amenable to valve reconstruction, and valve replacement can be carried out without time-consuming attempts at reconstruction. Reopening of the bridge and perfusion of the heart allows the reconstructed valve to be inspected before closure of the atrium to assess the operative result in the beating heart.

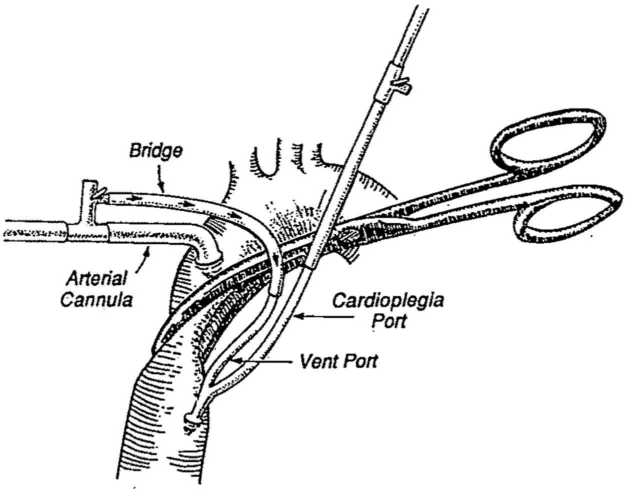

Fig 1. Bridge from arterial cannula to Y-cardioplegia cannula, which allows perfusion of the aortic root with aortic clamp in place.

The risk of systemic air embolization is eliminated with the aortic cross-clamp in place and there is no major ischemia of the myocardium with the coronary arteries being perfused before the delivery of cardioplegia.

The technique has been used in 10 patients; the valve was reconstructed in 4 and replaced in 6. Replacement was carried out in 2 patients with severe ischemic valve disease to minimize the ischemic time and in 4 patients because of severe calcific disease or ruptured papillary muscle.

The technique described is simple and safe, and may give the surgeon important information in the evaluation of the mitral valve before and after repair.

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Extended Use of Bilateral Internal Mammary Arteries for Coronary Artery Disease

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The internal mammary artery is the conduit of choice for coronary artery revascularization. Wide angulation between the left anterior descending coronary artery and obtuse marginal branches and the diseased segments of the coronary arteries can prevent optimal positioning of the left internal mammary artery for sequential anastomoses for revascularization. We describe a technique using a segment of the left internal mammary artery as a free graft sewn in an end-to-side fashion to the in situ left internal mammary artery. This approach has technical and physiological advantages over previously described techniques.

(Ann Thorac Surg 1990;49:1014–5)

Because of 10-year patency rates of approximately 90%, internal mammary arteries have become the conduit of choice for coronary artery revascularization [1]. Use of bilateral and sequential mammary arteries is technically more demanding than grafting with reversed saphenous vein; perpendicular side-to-side anastomoses are more difficult to construct and have less favorable flow characteristics. Some sequential anastomosis techniques require dependence of large areas of myocardium or of the entire coronary circulation on flow through one internal mammary artery [2]. We propose a method with technical and physiological advantages to achieve complete revascularization in select patients, particularly when the left anterior descending coronary artery and the obtuse marginal branches are separated by a wide angulation.

Technique

Three male patients aged 49 to 56 with class III angina pectoris were studied; 2 had three-vessel disease with severe proximal stenoses of the left anterior descending and circumflex coronary arteries, and the third had critical stenosis of the left main and right coronary arteries. The internal mammary arteries were dissected from below the bifurcation into superior epigastric and musculophrenic branches to near the junction with the subclavian artery. The arteries were divided distally and assessed for adequate flow. The pericardium was incised to the phrenic nerves bilaterally, and transverse incisions were made in the fascia of the mammary pedicles to provide maximal length. In all patients, attempts were made to provide complete revascularization and to construct mammary-to-coronary anastomoses immediately distal to the diseased area, allowing the maximal length of antegrade flow beyond the area of stenosis. Described techniques [2] for sequential positioning of the left internal mammary artery produced serious angulation between the obtuse marginal and left anterior descending arteries in all 3 patients.

The left internal mammary was measured to the appropriate length for the anastomosis to the left anterior descending artery and divided. The terminal segment of the left internal mammary artery, which measured 4 to 6 cm in length, was then anastomosed in an end-to-side fashion to the in situ left internal mammary artery at the level of the pericardial reflexion. After construction of the end-to-side mammary-to-mammary anastomosis, both limbs of the Y graft were assessed for adequate pulse flow. The right internal mammary artery was prepared for grafting to the distal right coronary artery; in 1 patient, it was divided proximally to produce a free graft to reach the posterior descending coronary artery.

Cardiopulmonary bypass was then instituted, and the patient was cooled to 26°C. The aorta was cross-clamped and blood cardioplegia was administered in the aortic root. Cross-clamp times were from 42 to 54 minutes. The in situ left internal mammary artery was anastomosed in an end-to-side fashion to the left anterior descending artery, and the free graft limb, to the obtuse marginal branch (Figs 1, 2).

Results

Postoperative recovery was uneventful in all 3 patients with no evidence of infarction or sternal healing difficulty. All patients remained free of angina and have had a negative stress thallium test.

Comment

The internal mammary artery has become the conduit of choice for coronary bypass grafting. Encouraging results suggesting similar long-term patencies with free internal mammary artery grafts [3] have expanded the possibilities and increased the number of patients who can receive complete revascularization with all grafts constructed using internal mammary arteries. Use of bilateral internal mammary arteries for complete revascularization can be

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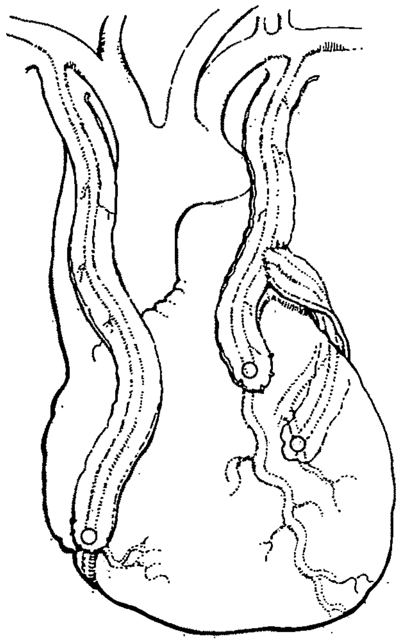

Fig 1. End-to-side left internal mammary artery Y graft with in situ right internal mammary artery.

difficult when the angle that separates the left anterior descending coronary artery and obtuse marginal branches is too wide for sequential use of the left internal mammary artery [4]. Techniques that require crossing of the right internal mammary artery anterior to the aorta may be disadvantageous if sternal opening is required [4]. Techniques requiring posterior placement of the right internal mammary artery through the transverse sinus have been associated with decreased patency rates [5]. Our technique of creation of a left internal mammary Y graft using a free segment of the distal left anterior descending has advantages including: (1) avoidance of angulation of the internal mammary artery graft to revascularize left anterior descending and obtuse marginal branches separated by wide angles; (2) assessment of flow of both limbs of the internal mammary artery graft before placement of the aortic cross-clamp; (3) increased number of end-to-side anastomoses and the ability to bridge diseased segments or perform intramyocardial connections without angulation; (4) placement of internal mammary artery grafts low and adjacent to the pericardial reflexion, away from the sternum in case another sternotomy is required; (5) easier options for complete revascularization leaving the right internal mammary artery for revascularization of the right coronary artery and its branches; (6) complete revascularization using flow from two internal mammary arteries versus "wrap-around" techniques with all flow dependent on one internal mammary artery; (7) use of the larger parts of the internal mammary artery with shorter grafts, both of which have been associated with improved flow through the internal mammary graft; and (8) expanded use of one internal mammary artery when bilateral internal mammary artery use is considered inadvisable because of diabetes mellitus, previous mastectomy, irradiation, or other reasons.

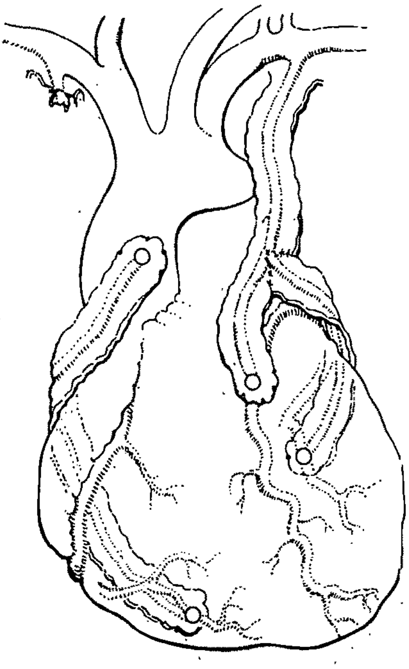

Fig 2. Left internal mammary artery Y graft with right internal mammary artery free graft.

This technique is simple and flexible, and it can be used in patients with more advanced disease as are more commonly seen since the advent of angioplasty. We have used this technique only in patients in whom saphenous vein was not available, and we thus recommend cautious extension of its use in patients with diffuse disease. The short-term and long-term patency of mammary-to-mammary anastomoses is unproven, and patients should be selected with high-grade stenoses in both the left anterior descending and circumflex arteries to avoid a potential steal syndrome associated with a Y graft configuration.

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Individual Ligation Technique for Lower Lobe Lobectomy

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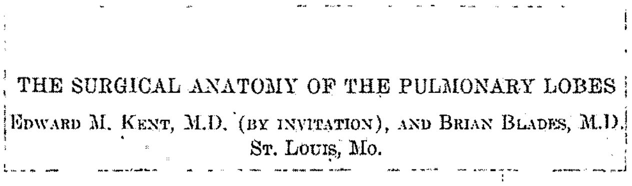
The classic description of the individual ligation technique for lower lobectomy was published in 1940. Doctor Brian Blades and Dr Edward Kent clearly described the anatomy of the bronchus, pulmonary artery, and vein to both the right and left lower lobes based on their dissection of 83 fresh cadaver lungs. They also provided a detailed description of their recommended technique for dissection, transection, and closure of each major structure. This was an important departure from the previously described mass ligation techniques. This cadaver experience was effectively transferred to the operating room. Kent and Blades reported on 18 patients who underwent lower lobectomy without mortality or the dreaded complication of "putrid empyema." They highlighted several basic thoracic surgical principles necessary for successful lower lobectomy that are as valid today as they were then. This landmark report provided the impetus for thoracic surgeons to accept lobectomy as a safe and standard anatomical resection.

(Ann Thorac Surg 1990;49:1016–8)



THE SURGICAL ANATOMY OF THE PULMONARY LOBES

EDWARD M. KENT, M.D. (BY INVITATION), AND BRIAN BLADES, M.D.
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Doctor Brian Blades and Dr Edward M. Kent presented their classic paper on the individual ligation technique for lower lobectomy at the 23rd Annual Meeting of the American Association for Thoracic Surgery at Cleveland, Ohio, in July 1940. This work was from the chest service of Barnes Hospital and Washington University School of Medicine, St. Louis, Missouri. Doctor Kent had been a resident in Thoracic Surgery at the Connecticut State Sanatoria from 1937 to 1938, and at the time of this effort, he was a Fellow in Thoracic Surgery on the chest service of Barnes Hospital. Doctor Blades had completed a fellowship in surgery at Barnes Hospital in 1938 and was an instructor of clinical surgery at the Washington University School of Medicine. A publication of such importance today would probably have from five to seven co-authors, and the last name would be the chief of the department. It is surprising to me that the name of Dr Evarts Graham is not on the manuscript, and I have no idea as to why he was not the senior author.

Lobectomy by individual ligation was beginning to be done in other centers, but this report brought the technique to a precise focus by combining a detailed anatomical description with a successful clinical result in 18 patients. Complete familiarity with the bronchus, pulmonary artery, and vein to the lower lobes was accomplished by the dissection of both lungs in 83 fresh cadavers. Blades and Kent very correctly noted that it was not possible to transpose safe operative techniques from that of an embalmed cadaver to a living patient, and the fresh specimens provided a more realistic technical exercise. This report described only the anatomy of the lower lobes, but Blades and Kent indicated that description of the anatomy and techniques for upper and middle lobectomy would be forthcoming. This occurred at the next meeting of the American Association for Thoracic Surgery in 1941, when Drs Kent and Blades presented their paper on the surgical anatomy of the pulmonary lobes [1]. Their observations at this time were based on the dissection of 227 lungs, and their clinical experience, on 27 lobectomies.

The first portion of the report describes the anatomy and recommended techniques of dissection, whereas the last portion is devoted to the clinical results. The interlobar fissures were described as complete, incomplete, or absent, and this terminology is commonly used today. It was recommended that the fissures be separated carefully along their anatomical plane, and defects in the lung parenchyma were closed with interrupted catgut suture. Vigorous blunt dissection of the fissure was condemned to avoid problems of hemorrhage and fistula.

The pulmonary artery to the lower lobe was described basically as a single trunk (Fig 1), but it was clearly recognized that the dorsal lobe or superior segmental artery often arose as a major branch and would have to be individually ligated. The arteries to the middle lobe were not anatomically defined; but reference was made to their presence, and they did not pose a problem in ligation of the main artery to the lower lobes. The Kelly clamp was recommended as the instrument of choice for dissection of the artery, vein, and bronchus. There was no mention of identifying the proper plane of dissection around the pulmonary artery nor the techniques of isolating the vessels using scissors, small dissecting sponges, or angled clamps. Inflammatory lymph nodes were thought to be more of a problem when isolating the bronchus; and if they presented any difficulty in freeing the lower lobe

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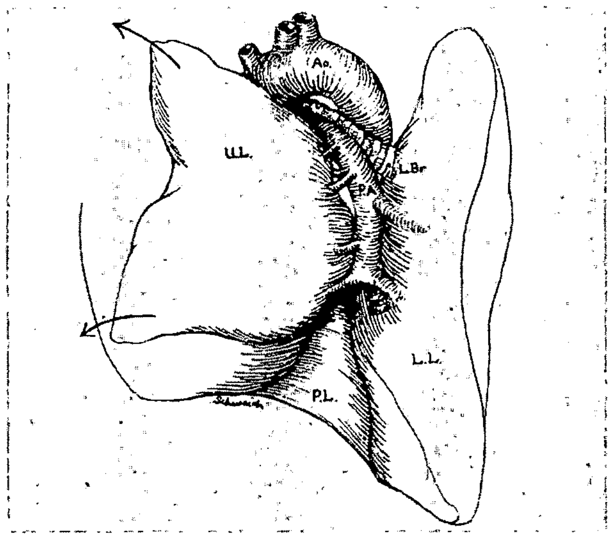

Fig 1. Diagrammatic illustration of pattern of left pulmonary artery as seen in the interlobar fissure. Note the several short branches to the upper lobe as the pulmonary artery passes around and behind upper lobe bronchus; the arterial branch to the superior division of the left lower lobe arises at a level above the point of origin of the artery to the lingula in this specimen. Ao., *aorta;* U.L., *upper lobe;* L.L., *lower lobe;* L.Br., *branch bronchus to superior division left lower lobe;* P.A., *left pulmonary artery; and* P.L., *inferior pulmonary ligament. (Reprinted from [1] by permission of C.V. Mosby Co.)*

artery, which is still frequently the case, they were excised.

The pulmonary artery was the first structure to be isolated and transected as it was determined to be the most accessible. Ligation of the main trunk of the pulmonary artery to both lower lobes would frequently include the branch to the superior segment of the lower lobe. It was recommended that the superior segmental artery be identified before the main trunk to the lower lobe was divided; and if it was a prominent or separate branch, it should be individually ligated and transected. There was no mention of the possibility of encroachment upon a branch of the pulmonary artery to the middle lobe when the main trunk to the lower lobe was ligated. The ligation technique consisted of a silk ligature around the major vessel to the lower lobe and tying it as proximal as possible. A clamp was then placed distally that included some lung tissue, and the artery was transected at the edge of the clamp. The distal artery stump was suture-ligated to prevent hemorrhage and after this, the proximal artery stump was grasped with a clamp and a second silk ligature was applied. Fragility of the pulmonary artery was mentioned as a possible complication, and clamps were always removed as quickly as possible. No mention was made of the possibility of placing a double ligature before transection of the artery or the use of a proximal suture ligature. If the ascending artery to the posterior segment of the right upper lobe was encountered during the dissection, it was simply ligated and transected as it was believed that no ischemia to the upper lobe would result if this artery was sacrificed. There was no discussion given that a more complete dissection might preserve this branch.

The pulmonary veins did not present a particular problem, but a special point was made about a small venous tributary that was frequently present in the superior portion of the inferior pulmonary ligament, and special care regarding its dissection and ligation was required. A single ligature was used to occlude the entire pulmonary vein, and no consideration was given to the possibility that the ligature might roll off or to the use of a double ligation technique. It was observed that the right inferior pulmonary vein could be exceedingly short and that the left lower lobe vein was more easily managed in a common ligature.

The superior segment of the lower lobe was described as a "lobe within a lobe," and a detailed discussion of the variations in anatomical origin (Fig 2) of the dorsal lobe bronchus was made. The length of each lower lobe bronchus was carefully measured; the average length of the right lower lobe bronchus was 2.0 cm, and that of the left lower lobe bronchus, 2.2 cm. Length of the bronchial stump was important to ensure that adequate closure could be obtained without compromising the lumen of the remaining lobar bronchi. Kent and Blades stressed that extreme care must be taken when the lower lobe bronchus was transected and closed to avoid partial occlusion of the right middle lobe bronchus. It was specifically noted that if the dorsal lobe bronchus arose above the origin of the middle lobe bronchus, then individual bronchial ligation should be done. This is a technical point that remains in our thoracic surgical practice today. Although careful

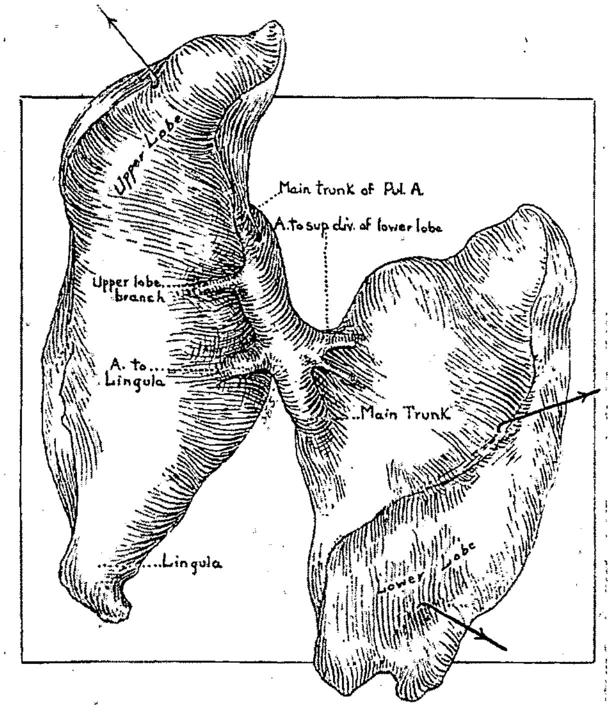

Fig 2. Diagrammatic illustration of distribution of left pulmonary artery as it lies deep in the left interlobar fissure; in this specimen the arterial supply to the superior division of the lower lobe is double and the point of origin is opposite that of the arterial branch to the lingula. (Reprinted from [1] by permission of C.V. Mosby Co.)

attention was used in describing the dorsal lobe bronchus, no mention is made of the basal segmental anatomy of the lower lobe. This is somewhat surprising, as 2 patients in this series had their lingula resected at the time of the left lower lobectomy and Churchill and Belsey [2] had reported segmental pneumonectomy for bronchiectasis in 1939.

The bronchus was transected between two Kelly clamps, and the proximal end was "carbolized" to minimize infection. Two mattress transfixion sutures of silk or catgut were placed, the bronchial stump was then closed with sutures of D silk or catgut, and the stump was covered with mediastinal tissue if possible. Adjacent portions of the middle lobe were also used to cover the stump in some of the patients.

Ten patients underwent successful lower lobectomy for bronchiectasis (9 patients) and a mixed tumor of the bronchus (1 patient). The patients were young, aged 10 to 40 years, and the hospital stay was from 18 to 42 days (average, 30 days). Two patients are worthy of mention in that 1 had been operated on previously for an empyema, and the other had a prior surgical drainage of a lung abscess. Even with our current anesthetic and operative techniques, pulmonary resection for chronic inflammatory disease can be demanding and difficult. The successful result in this group of patients certainly attests to the fortitude and expertise of these surgeons. Chest tubes were placed anteriorly and posteriorly, and 4 patients drained only straw-colored fluid and did not develop an empyema. However, empyemas did develop in 6 patients, but they were described as not being of the putrid type. Five patients had a bronchial leak 2 to 3 weeks after the lobectomy. The leaks were small, detected only by the irrigation of the residual space through the chest catheters, which was done to avoid the development of a putrid empyema. The bronchial fistulas were small, as a substantial exchange of air was not detected through the thoracostomy tubes, and they may well have been leaks from lung tissue along an incomplete fissure.

Two patients had resection of their lingula in conjunction with left lower lobectomy, and 1 patient had a middle lobectomy with the right lower lobectomy. In the latter instance, the middle lobe was removed by the clamp and transect technique, and one must speculate why individual ligation of the structures to the middle lobe was not carried out. In a footnote to the table describing the 10 patients, the authors report an additional 8 patients that underwent successful lobectomy for bronchiectasis (6 patients), lung abscess (1 patient), and a foreign body (1). After operation, 1 patient died following surgery of a brain abscess, and postoperative empyema occurred in 9 of the 18 patients.

Other thoracic surgeons were performing individual ligation of hilar structures for lobectomy at the same time. Churchill and Klopstock [3], in their report on lobectomy for pulmonary tuberculosis, stated that lobectomy by the individual ligation technique had been performed in their hospital since 1936 and discussants of the paper reported on various lobectomies using similar techniques. Doctor E. F. Butler reported on the removal of ten lobes in 8 patients without a death. Doctor Harold Brunn, an advocate of the Shenstone snare for lobectomy, commented that he had operated on a number of patients for tumors and bronchiectasis using the technique of individual ligation. He believed that a cleaner bronchial stump could be obtained and avoidance of drainage of the pleural space was possible. Doctor John B. Flick made a most pertinent comment when he stated that "covering over the stumps with viable, healthy tissue will lessen the incidence of bronchial fistulas and of empyemas." He reported success with individual ligation in 2 patients, but also had failed with his dissection in several other patients and had to revert to the clamp technique. Doctor J. A. Moore discovered an artery going directly into an infected cyst in the lung. He was obviously dealing with an infected intralobar sequestration and was successful in both ligating the systemic artery and completing the lobectomy. Doctor Blades commented in his closing remarks that he had never seen such a case.

Several basic principles of thoracic surgery that have withstood the test of time and experience were brought out in this report [1]. The recommended sequence of dissection and transection of the hilar structures was the pulmonary artery first followed by the vein and bronchus, and this is the usual order in which most pulmonary resections are accomplished today. Kent and Blades recommended that preoperative bronchoscopy be done to define the bronchial anatomy. This basic principle is just as important today as it was then. The close proximity of the middle lobe orifice to the superior segmental bronchus was clearly recognized, and whether the bronchus is stapled or sewn, care must be taken to avoid middle lobe bronchial compromise. There was comment on the difficulty of freeing adhesions between the bronchus and the pulmonary vein, and fibrosis in this anatomical area can still present a technical difficulty. Other comments applicable to modern-day thoracic surgery include the fragility of the pulmonary vessels, the necessity to clearly identify the pulmonary artery to the superior segment of the lower lobe, and the vulnerability of pulmonary veins to accidental injury. Last, but certainly not least, a statement that "any bronchus stump which opens postoperatively presents a potentially serious complication" is just as true 50 years later.

One can only admire this landmark report by Kent and Blades on surgical technique and clinical result for lower lobectomy, which today we accept as routine. It is interesting to note that the lobe to be removed was "attacked" and each resection was obviously a challenging experience. I would have liked to hear the comments in the operating room as they were carrying out their first lobectomy using the individual ligation technique.

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Familial Achalasia in Children

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Familial achalasia is rare, especially in children. Five siblings with achalasia surgically treated at ages 5½ months to 19 years are reported. There is no history of consanguinity or of vertical transmission.

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This report is concerned with 5 sibling children (3 boys and 2 girls) with achalasia of the esophagus who were operated on between 1964 and 1987 at the ages of 5½ months, 2, 3, 9, and 19 years. They are from an Iowa family with 9 children, in which the father is a farmer and the mother is a registered nurse. There is no history of consanguinity or vertical transmission.

Case Reports

Patient 1

John, age 2 years, was cared for by Dr Homer Skinner of Carroll, Iowa. The child had swallowing difficulty, especially with solid food, since birth. He had pneumonia twice during his first year of life. Physical examination was unremarkable, but an esophagram disclosed wide dilatation with distal narrowing. Transabdominal esophagocardiomyotomy was performed in October 1964 by Dr Skinner with an excellent result. An esophagram 6½ years later showed an apparently normal esophagus. He is now 27 years old and has no swallowing or respiratory symptoms, although he is hypertensive.

Patient 2

Jamie, age 3 years, was hospitalized by a pediatrician, having been referred with a diagnosis of achalasia after an esophagram. The child had a 6-month history of poor appetite, spitting up after eating, and nocturnal cough. He had been treated for allergy with little improvement. In December 1972, esophagoscopy was performed followed by transthoracic modified Heller esophagomyotomy. Postoperative course was smooth, and the child has had no further respiratory or swallowing problems. An esophagram on the sixth postoperative day disclosed considerable decrease in the size of the esophagus. At 19 years of age, he is hypertensive but has no respiratory or gastroenteric symptoms.

Patient 3

Jerret was 9 years old when hospitalized by a pediatrician with a diagnosis of achalasia. Seven months earlier cough developed, especially at night. Because exposure to barn or grain dust seemed to aggravate the cough, allergies were suspected. Unlike his brothers, he gave no history of difficulty with swallowing or of vomiting, although it had been noted that his pillow and pajama tops were often wet in the morning. Ten days before hospitalization, he had a respiratory infection, and a patchy infiltrate was noted in the right lower lung field. Because of the family history, his family physician suggested an esophagram, which demonstrated achalasia. Jerret's mother, a nurse, was chagrined that she had not considered this possibility earlier but was misled by the predominant respiratory symptoms. A transthoracic modified Heller procedure was performed in June 1975 with a satisfactory result. One month later, an esophagram demonstrated decrease in the esophageal caliber and postsurgical deformity of the distal esophagus. Jerret has remained asymptomatic except that at age 23 years he has also become hypertensive.

After Jerret's operation, the remaining 4 children born at that time were examined with esophagrams of which 3 showed negative results. Jody (patient 5), a 9-year-old girl, had a moderately suspicious radiographic examination but was asymptomatic. She was reexamined 1 year later at which time she was asymptomatic, and the moderate esophageal dilatation had decreased.

Patient 4

Jennifer was 5½ months old when referred with a diagnosis of achalasia. Two weeks earlier, she had been started on pureed and solid foods, and it was noted that she would spit up food at night. She began having noisy respirations at night, similar to that experienced by her brothers. She also had increased regurgitation of saliva. Esophagram demonstrated a dilated esophagus with distal tapering.

Examination was remarkable only in that the child spit up rather large amounts of saliva during the examination, even while sitting up. In August 1977, a transthoracic modified Heller procedure was done. The postoperative course was smooth, and esophagram on the eighth postoperative day demonstrated decrease in esophageal dilatation with slight postoperative irregularity of the distal esophagus. On reexamination 2½ months later, she was thriving and had gained 0.9 kg. She had no cough, breathing difficulty, or regurgitation.

She was seen again on February 1, 1979, at the age of 23 months after examination by her family physician because of a recent cough. Chest roentgenogram disclosed a small infiltrate in the right paracardiac region. An esophagram was reported as showing dilatation of the distal esophagus plus the presence of a foreign body filling defect. On examination, the child weighed 12.7 kg. Her mother reported that she was a fair eater and had no apparent problem with swallowing. A review of the esophagram

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suggested the possibility of a pineapple chunk, which the child had eaten the preceding night, as responsible for the filling defect. Otherwise, the x-ray films appeared essentially unchanged from the earlier postoperative films, with no dilatation above the myotomy site.

Esophagogastroscopy was performed and there was no evidence of esophagitis, stricture, reflux, or foreign body. The esophagogastric junction tended to open slightly as the scope approached the junction, which the scope readily traversed. A repeated barium swallow demonstrated ready passage of barium into the stomach, with minimal delay of a small amount of barium. Now at 12 years of age, she is normal except for hypertension and has no swallowing problems.

Patient 5

Jody, a 19-year-old college student, was referred by a gastroenterologist with a diagnosis of achalasia. At the age of 9 years, she had an esophagram that suggested esophageal dilatation but was asymptomatic. On repeat radiographic examination 6 months later, the esophagus was thought to be improved. She remained asymptomatic until 7 months before referral at 19 years of age. At that time, she began to notice regurgitation of food when she would lie down. An esophagram demonstrated achalasia. She was referred to a gastroenterologist who performed a pneumatic dilation of the esophagus. Temporary improvement followed; she had no recurrence of regurgitation but did have an increasing sensation of food sticking in the esophagus and a nonproductive cough. The patient was slender, and physical examination was unremarkable. Her esophagrams demonstrated a moderately dilated esophagus with a tapered distal end. In May 1987, a transthoracic modified Heller esophagomyotomy was performed. She recovered uneventfully and reported a distinct improvement in swallowing. On reexamination 4 months later, she was asymptomatic and her cough had disappeared. A postoperative esophagram displayed a distinct decrease in esophageal dilatation. Now at age 21 years, she is asymptomatic. She was normotensive at the time of her operation.

At the time of operation on patient 5, the patients' mother reported that 4 of the children with achalasia had developed hypertension, while none of the children with no achalasia had become hypertensive. In a recent communication with the patients' mother, all children with achalasia are asymptomatic with respect to their respiratory and gastrointestinal tracts. Also, 3 of the children with no achalasia are likewise asymptomatic. Swallowing symptoms that could be related to achalasia have developed in one male sibling now in his late twenties, but he refuses to have appropriate diagnostic studies. There is no history of alacrima or hypocorticism. There is also no history of achalasia in previous generations on either side of the family. The association of hypertension with achalasia in this family is probably not significant. The father is hypertensive, as are others on his side of the family.

All 5 siblings with achalasia were treated surgically, 1 with transabdominal esophagocardiomyotomy, and 4, with transthoracic modified Heller esophagomyotomy in which particular care was taken to extend the myotomy onto the cardia 0.5 cm or less. The last patient was treated with esophageal dilation preoperatively with transient decrease in symptoms.

Comment

Achalasia of the esophagus is an uncommon condition and is quite rare in children, especially in infants. Nevertheless, a number of reports from around the world demonstrate a familial occurrence of achalasia, with multiple siblings, both adult and children, being involved.

The reasons for this familial occurrence have been speculated on, with environmental, infectious, and genetic factors being considered. (In a personal communication in 1975, Dr Hans Zellweger, Professor of Pediatrics at the University of Iowa and a geneticist, expressed the opinion that these reports of several affected siblings supported the assumption that achalasia occurs as an autosomal recessive trait in rare instances.) Several other reports [1–6] have made similar suggestions. Stein and Knauer [4] interpret the development of achalasia in cases of vertical transmission, the lack of consistent vertical transmission, and the finding of cases in families with consanguinity as suggestive of an autosomal recessive type of inheritance. Westley and colleagues [3] believed that the lack of evidence of vertical transmission and the occurrence of achalasia in children of both sexes and in multiple members of an extended family constituted strong evidence that a recessive gene was responsible for infantile achalasia. Mayberry and Atkinson [7] studied 1,012 first-degree relatives of 167 achalasia patients of which 14 were said to have dysphagia and 2 were reported to have achalasia. However, Mayberry and Atkinson were unable to confirm the diagnosis of achalasia in any of the relatives and concluded that adult achalasia is not inherited in an autosomal recessive fashion. Familial achalasia has also been noted in several families with a high degree of inbreeding [2–5, 8].

In 1962, Tyce and Brough [9] reported multiple diseases inherited in three generations of a family. Of the 9 members of the third generation of this family, 7 were mentally retarded, and 4 brothers demonstrated achalasia, mental retardation, speech disorder, and neurological disease with cerebellar components. This is believed to be the first reported instance of familial achalasia. In 1965, Thibert and associates [10] provided the first description of familial achalasia in children with reports of two separate groups of male siblings. One group included 4-year-old and 16-year-old brothers, and the other, 11-year-old and 14-year-old brothers. Cloud and colleagues [11] studied several children with achalasia, including 4 Apache Indian brothers. A Mexican family that immigrated to California had 3 children with achalasia, and a fourth who died in Mexico without medical attention may also have had this condition [12, 13]. Additional reports of children with familial achalasia came from Turkey [14] and Hungary [15].

Bosher and Shaw [16] recorded the discovery and treatment of 2 brothers with achalasia in early childhood. Their review of the world literature in 1981 disclosed only 33 cases of familial achalasia including their own. Their report, however, did not include those noted above [9–11].

It is important to appreciate the clinical significance of pulmonary symptoms in the diagnosis of achalasia in children. Respiratory symptoms including cough, wheeze, and pneumonia were prominent in all children with achalasia in the family on whom the present report is based. The importance of pulmonary symptoms in the histories of children with achalasia has been emphasized by Vaughan and Williams [17] and by Schultz [18].

A wide variety of syndromes associated with achalasia has been reported and has been reviewed in detail by Feldman [19]. These include Sjögren's syndrome [20, 21] and the so-called triple-A syndrome consisting of achalasia, adrenal insufficiency, and alacrima [8, 22–24]. Various forms of mental retardation in association with achalasia have been reported [8, 9, 13, 24], as well as congenital cholinergic dysfunction in identical infant twins [25].

Zimmerman and Rosenweig [6] have commented that approximately two thirds of reported cases of familial achalasia have occurred in children under the age of 15 years. Yet, achalasia occurs in children less than 5% of the time [1, 26, 27].

In children with achalasia, the treatment is preferably surgical [27–30]. The results of dilation in children are less satisfactory than in adults [27], and dilation is characterized as a temporizing, nondefinitive procedure by Polk and Burford [29].

The performance of a modified Heller esophagocardiomyotomy in achalasia in children achieves approximately 70% to 85% good long-term results [27, 29, 31]. Careful attention to the prevention of disturbance of the esophagogastric junction and limitation of the extension of the myotomy onto the gastric cardia should reduce the incidence of postoperative reflux [29, 32, 33]. Although the modified Heller myotomy is the procedure of choice in the management of achalasia in children, some surgeons have added a Nissen fundoplication [34] or a modified Belsey mark IV fundoplication [35] when there was an increased risk of gastroesophageal reflux.

Conclusion

Achalasia, although seen at all ages, is an uncommon condition and is rare in children. In most cases there is no family history, and it occurs as an isolated entity of unknown etiology. However, several reports from around the world indicate a familial occurrence of achalasia. It has been suggested that it may be transmitted as an autosomal recessive trait; however, environmental and infectious etiologies have also been suggested with little to support these theories, although Chagas disease does cause a somewhat similar esophageal lesion. In the family of 9 children reported here, 5 siblings, aged 5½ months to 19 years, were treated with a modified Heller esophagomyotomy with excellent results. A sixth sibling has symptoms that are compatible with achalasia, but has refused investigation. In each child reported here, respiratory symptoms such as cough, wheeze, and pneumonia were prominent and were sometimes the initial symptoms.

I am indebted to Dr Homer Skinner for furnishing the information regarding patient 1.

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The Cardiac Surgery Program of the National Heart, Lung, and Blood Institute*

Invited Letter to the Editor:

Thank you for giving me the opportunity to comment on and explain the recent decision to phase out the cardiac surgery program of the National Heart, Lung, and Blood Institute (NHLBI).

From a historical perspective, the cardiac surgery program of the NHLBI has made many outstanding contributions to the fields of clinical surgery and surgery research. In addition, the cardiac surgery program was essential and integral to the research program of the Institute's Cardiology Branch, by providing surgical support and follow-up to the patients admitted in the Cardiology Branch program. I should also underscore that the cardiac surgery program has had a tradition of high quality training. This was well recognized in a letter I recently received from Dr W. G. Rainer, President of The Society of Thoracic Surgeons. He said, "Many of our leading thoracic surgeons have been through fellowship training at (NHLBI)."

Indeed, this aspect of the program is a tribute to its founder, Dr Andrew G. Morrow (1953–1982), and to Dr Richard E. Clark (1983–present), who followed Dr Morrow. For example, of the 103 clinical associates who were in the program between 1953 and 1979 for whom follow-up information is available, 61 are now tenured professors. Of these 61 tenured professors, 37 are division heads, 15 are department chairmen, and 3 are chief administrators. The other 42 clinical associates are now in private practice. These are remarkable statistics of which we are fully aware. In fact, this was one of the reasons that made it so difficult to reach the decision to phase out the cardiac surgery program.

Nevertheless, several important factors led to this decision. (Parenthetically, I should emphasize that my decision was discussed with and supported by the Acting Director, National Institutes of Health [NIH], and the Assistant Secretary for Health of the Department of Health and Human Services.)

One of the key factors considered is the number and nature of the surgical procedures recently done at the Institute as shown in Tables 1 and 2. These procedures represent the workload of a staff of four cardiac surgeons, of whom only two were operating. Thus, even though the Institute has supported its cardiac surgery program for its research contribution, rather than for clinical care per se, this workload is by common standards rather modest, particularly in view of the magnitude of the total resources required to support it.

This leads us to the next element that was considered—the cost of performing cardiac surgery at the NIH. Those who know the way NIH functions are well aware that the cost of any clinical research program has two components: the cost of the research per se, which is paid directly by the Institute doing the research, and the clinical care costs, which are paid by the Institute to the Clinical Center. Table 3 shows the Clinical Center charges to the NHLBI for its six clinical research programs (Cardiology, Pulmonary, Hematology, Hypertension, Molecular Disease, and Cardiac Surgery) and the charge for surgery alone.

Obviously, phasing out the cardiac surgery program does not result in a "savings" equal to its current costs in the Clinical Center. Most of these expenditures represent fixed Clinical Center costs that cannot be eliminated. However, those same resources can be committed to activities other than surgery. For instance, Clinical Center beds (which are in limited supply) and the highly talented nurses serving cardiac surgery are needed to support high-priority clinical research programs within the Institute.

Table 1.

Type	1986	1987	1988	1989 (6 mo)	Total
	Number of Cases				
Elective first operations	118	131	145	77	471
Emergencies	13	7	11	4	35
Redo/reops	29	23	38	9	99
Total	160	161	194	90	605

Another significant factor is the changing nature of cardiology research. It is widely known that the cardiac surgery program has conducted a productive research program since its inception. Its work in the areas of hypertrophic cardiomyopathy and valve replacement procedures has been pioneering and outstanding. However, today these procedures have now been accepted as standard clinical practice and are routinely performed in many institutions in this country, some of them in the Bethesda area. Although the Institute's Cardiology Branch will continue and even strengthen its clinical research in some areas, and continue to admit the same number of patients, the need for direct surgery support for the cardiology research program is decreasing. For example, the Cardiology Branch has decided not to continue clinical research on angioplasty and other interventional therapy, such as atherectomy procedures and laser angioplasty, but will maintain its diagnostic and research catheterization activities. Concurrently, the Cardiology Branch will expand its efforts in the use of molecular and cellular biology approaches to study the pathogenesis and treatment of heart disease. Whenever surgery for patients admitted to the cardiology clinical research program is needed, it will be performed at one of our nearby hospitals with outstanding surgical experience.

A final critical factor that we considered was the retirement of one of the two operating surgeons in the Surgery Branch. Because of this surgeon's unique experience in the surgical treatment of hypertrophic cardiomyopathy, it became clear that, without this experience, our cardiac surgery program would no longer be any different from other programs. Moreover, we could not keep the program going with only one operating surgeon. The current chief of the program interviewed a number of potential candidates, of whom one or two appeared very likely to meet our requirements. Yet even with the recruitment of one of these candidates, it still would not be possible to reestablish a long-term stable situation. The federal pay structure remains an obstacle to stability, particularly from the viewpoint of a young person such as the most suitable candidate. Thus, in considering this factor along with all the others mentioned herein, I believe that it is to the best advantage to the Institute to phase out the cardiac surgery program and arrange for off-site cardiac surgery.

Cardiac surgery is now a mature discipline, and high-quality cardiac surgery is widely available throughout the United States.

Table 2.

Type of Cases (3½yr)	No.	%
Coronary graft	170	28
Cardiomyopathies	119	20
Valve (only or with other procedures)	262	43
Congenital	47	8
Other	7	1
Total	605	100

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* By prior agreement, this letter is being published both in *The Annals of Thoracic Surgery* and in *The Journal of Thoracic and Cardiovascular Surgery*.

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Table 3.

	Clinical Care Costs		
Year	Six Clinical Programs ($)	Cardiac Surgery ($)	Percent
1986	20,699,058	6,418,964	31.0
1987	23,825,894	8,240,562	34.6
1988	27,503,246	10,611,389	38.6
1989 (6 mo)	13,274,699	5,247,521	39.5
Total	$85,302,897	$30,518,436	35.8

Thus, the need for a clinical unit at the NIH is much less now than it was during the pioneering period. I recognize the need for vigorous research in cardiac surgery and realize that new advances can bring immense benefits to the American people, as has been the case during the past four decades. Through its extramural grant program, the NHLBI continues to support the research activities of cardiac surgical principal investigators all over the country. By way of this letter, I wish to strongly reaffirm my support and endorsement of sound, innovative research activities in cardiac surgery.

As tangible evidence of my support for cardiac surgical research and my expectation of future advances, I plan to continue the cardiac surgical fellowship program for at least 5 years. Of course, I am fully aware that the surgery academic community has always considered the cardiac surgery program of the Institute as a place to send young surgeons for research training. In the past, we have enrolled four new clinical associate surgeons each year for a 2-year minimum stay. I have committed the continuation of this program for 5 years, after which it will be evaluated. The surgical associates will devote full time to research in any of the 16 NHLBI laboratories and/or branches. If successful, that is, if young surgeons use this program, it will be continued. An announcement for this program has been prepared and immediately follows this letter.

The Institute also has an ongoing commitment to its patients, past and present. We will continue to evaluate potential surgery patients in the cardiology program and will provide medical treatment at the Clinical Center. We will also continue the outpatient clinic for patients who have previously had cardiac surgery at the Clinical Center. Former and new patients needing surgery will be referred to other local surgery facilities whose staff will be working closely with the clinical and research staff of our Cardiology Branch. All costs that are not covered by the patient's insurance will be underwritten by the Institute; there will continue to be no cost to the patient.

I hope this letter explains the background and the rationale behind the decision to phase out the Institute's cardiac surgery program. As I said earlier, it was a very difficult decision to make, but I believe an inevitable one. The problems associated with this decision are greatly and favorably balanced by the opportunity it creates to expand existing programs, such as in cardiology and hematology, and to create new ones that will help the Institute remain at the forefront of biomedical research.

One last point is important. Some have interpreted the phasing out of the *intramural* cardiac surgery program of the NHLBI as evidence of a declining interest on the part of the NHLBI in supporting *extramural* research related to cardiothoracic surgery. Such is in no way the case. The NHLBI remains as receptive as ever to applications for support of research relevant to cardiothoracic surgery.

Claude Lenfant, MD

Director, NHLBI
National Institutes of Health
Bethesda, MD 20892

The following information is provided as a supplement to Dr Lenfant's letter.

National Heart, Lung, and Blood Institute Surgical Associate Program

The National Heart, Lung, and Blood Institute (NHLBI) directs the nation's attack on diseases of the heart, blood vessels, lungs, and blood. It is one of the 13 institutes at the National Institutes of Health (NIH) located in Bethesda, Maryland, a suburb of Washington, DC. The NHLBI's intramural research program is one of the largest at the NIH and includes 16 laboratories and branches, with researchers working in nearly every discipline of fundamental and clinical study. As a part of the NIH, NHLBI intramural researchers benefit from the atmosphere of intellectual stimulation and exchange, as well as numerous shared resources such as a 500-bed research hospital, various research support services, and the world's largest repository of medical literature.

The Surgical Associate Program

The NHLBI Surgical Associate Program offers an opportunity for young physicians to obtain training with leading investigators at the forefront of biomedical research. The Surgical Associate devotes full time to clinical and/or laboratory studies with no patient care responsibilities other than those that may be directly related to the research.

Current research areas include the biology of experimental and clinical arteriosclerosis and its manifestations; the pathophysiology of hypertensive vascular disease; structure and function of the lung; physiologic and pharmacologic aspects of heart, blood, and lung diseases; and a broad program of basic research including angiogenesis, growth factor and endothelial cell biology, cardiac energy metabolism, ischemia, and molecular cardiology.

Eligibility

An applicant should be (a) an MD who has completed a minimum of 2 core years of training in a surgical residency program approved by the Council on Medical Education; (b) preparing for a full-time academic career in cardiothoracic surgery; and (c) a US citizen or a resident alien eligible for citizenship within 4 years.

Support

The annual salary is $37,000 to $41,000. The initial appointment is for 2 years, with the possibility of extension. Additional benefits include health and life insurance subsidy, government retirement, annual vacation and sick leave, travel and movement of household effects to the NIH, and educational training. There is no requirement for payback.

How to Apply

For additional information on research opportunities and application forms, interested individuals should contact:

Dr Edward D. Korn
Director, Division of Intramural Research
National Heart, Lung, and Blood Institute
Building 10, Room 7N214
Bethesda, MD 20892

Applications are due November 30, 1990, for appointments beginning July 1991. Candidates will be notified by February 1, 1991.

Operation for Hypertrophic Cardiomyopathy

To the Editor:

We read with interest the excellent article by Walker and associates [1] describing their experience with the surgical management of hypertrophic obstructive cardiomyopathy. Similarly, the invited commentary on that paper by Charles MacIntosh of the National Institutes of Health group was informative. We would, however, suggest a note of caution in applying a "set" preoperative formula in at least some of these patients.

Over a 15-year period in a program involving 300 to 500 open cardiac cases per year, we have encountered 5 adults and older children (age range, 14 to 66 years) receiving operations for hypertrophic obstructive cardiomyopathy. In only 1 was this the only lesion producing left ventricular outflow obstruction, each of the others having either a subaortic diaphragm or valvar aortic stenosis [2, 3].

The presence of these additional lesions is not always obvious because of limitations of echocardiographic and angiographic assessment. Combined lesions can be treacherous in that drug therapy, such as propranolol and verapamil, can cause marked deterioration as occurred in 2 of our patients necessitating urgent operation [4].

We would suggest that the surgeon strongly consider inspecting the left ventricular outflow tract and the mitral valve before making a final decision on how to deal with the problems presented by the individual patient.

With regard to questions of technique, the papers of Marrow and associates [5], Cooley and co-workers [6], and MacIntosh and colleagues [7] give the surgeon very satisfactory guides to the selection and performance of appropriate procedures.

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R. V. Bernstein, MD
A. Damle, MD
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Reply

To the Editor:

My colleagues and I thank Dr Browdie and colleagues for their kind remarks and interesting observation. Fortunately, we have not had the experience of finding that hypertrophic obstructive cardiomyopathy was an incomplete cause for the left ventricular outflow tract obstruction present in any of our patients. In general, we believe that modern investigative cardiological techniques including left ventricular angiography and echocardiography should render this event unlikely in adults, but it is certainly the case that any diagnosis should be verified by the surgeon at operation. In this regard, mitral valve replacement may offer an additional advantage over transaortic septectomy in the management of hypertrophic obstructive cardiomyopathy because, having resected the mitral valve, the surgeon can digitally evaluate the left ventricular outflow tract and aortic valve (as is our practice with all mitral valve replacement procedures). A transaortic approach, however, provides for only limited assessment of the ventricular aspect of the mitral valve anterior leaflet, so that a separate left atriotomy would be required to fully evaluate the valve.

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Cardiac Function With Glucose-Insulin-Potassium Solution

To the Editor:

We read with interest the article "Improved cardiac function with glucose-insulin-potassium after aortocoronary bypass grafting" by Gradinac and colleagues [1]. Their results demonstrate dramatic improvement with metabolic enhancement in a group of patients with severe left ventricular dysfunction after reperfusion of ischemic myocardium. Following the reports of Bormann [2], Lolley [3], and Oldfield [4] and their colleagues in 1985 and 1986, at the University of Nebraska Medical Center we began to administer preoperative infusions of glucose-insulin-potassium (GIK) in a select group of patients undergoing myocardial revascularization and/or valve replacement. It was felt that increasing the intracellular glycogen stores would allow these hearts to tolerate longer periods of ischemia by providing substrate for glycolytic pathways as well as during the critical period of early reperfusion.

In those patients with severe left ventricular dysfunction, particularly those requiring extensive procedures, we have also routinely used low-dose dobutamine infusions preoperatively, because of the demonstrated improvement of myocardial mitochondrial morphology and function [5]. With the combination of preoperative GIK and low-dose dobutamine, many patients in whom we would have predicted the need for postoperative balloon support have been weaned from cardiopulmonary bypass without difficulty. In most, continuation of low-dose dobutamine (3 to 5 μg/kg/min) was all the inotropic support that was required.

The results with *post*operative GIK cited by Gradinac and colleagues are impressive in their group of patients who were already in cardiogenic shock after myocardial revascularization. Our experience suggests that use of *pre*operative GIK, with or without low-dose dobutamine therapy, can be beneficial in patients in whom perioperative ventricular dysfunction can be predicted or anticipated. This may eliminate severe low output syndrome and obviate the need for mechanical ventricular assistance. It may also lower the dosages of required inotropes, and provide more rapid weaning, as demonstrated by the Houston group.

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Lary A. Robinson, MD

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References

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Reply

To the Editor:

My colleagues and I are grateful to Drs Moulton and Robinson for their comment on the utility of preoperative administration of glucose and insulin. It has surprised us as well that the idea of "glycogen loading" has not found wider acceptance after all the years it has been around. A large-scale, controlled clinical trial is badly needed at this point to confirm the encouraging initial observations.

One should point out that the mechanism for the beneficial effect of GIK and glycogen loading has not yet been identified with certainty. In a recently published study [1] on the effect of glycogen loading before hypothermic, ischemic arrest of isolated rabbit hearts, we found not only significantly improved functional recovery after 12 hours of ischemia but also a direct correlation between the tissue contents of glycogen and energy-rich phosphates. Quite obviously, more work is needed before we know what happens at a molecular level, but the results are promising if one thinks of extending the ex vivo viability of hearts destined for cardiac transplantation.

Heinrich Taegtmeyer, MD, DPhil

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Reference

1. McElroy DD, Walker WE, Taegtmeyer H. Glycogen loading improves left ventricular function of the rabbit heart after hypothermic ischemic arrest. J Appl Cardiol 1989;4:455–65.

Lipoid Pneumonia After Laryngectomy Mimicking Pulmonary Malignancy

To the Editor:

Exogenous lipoid pneumonia resulting from foreign body reaction to inhaled mineral, animal, or vegetable oils poses interesting diagnostic problems for the clinician, the radiologist, and the thoracic surgeon. This condition was first reported by Laughlen [1] in 1925 and subsequently extensively documented by Pinkerton [2] and Ikeda [3], who described the nonspecific clinical and roentgenological features of this condition, which often resemble those of other diseases of the lung, particularly bronchogenic carcinoma. The increasing incidence of lung cancer and its association with upper aerodigestive malignancies has stressed the existence of common risk factors, identifying a definite group of patients at high risk. Consequently, when a pulmonary mass is detected in this group of patients, it is imperative to rule out malignancy.

Two patients who had previously undergone laryngectomy for carcinoma were referred to our department for evaluation of a pulmonary mass. The first patient was referred 3 years after laryngectomy; chest roentgenogram and computed tomographic scan showed two lesions localized in the middle and left lower lobes. Fiberoptic bronchoscopy with bronchoalveolar lavage and transthoracic fine needle aspiration gave negative results. The patient was placed on a course of broad-spectrum antibiotics, which resulted in the complete regression of the lesion in the left lower lobe. Surgical exploration of the right pulmonary lesion was deemed necessary for diagnosis and treatment of possible malignancy. At operation a wedge resection of the area of consolidation was performed.

The second patient was evaluated 6 years after laryngectomy because of dyspnea, fever, hemoptysis, and left chest pain. Three years earlier two pulmonary metastases had been removed. At the time of presentation radiological work-up showed the presence of an area of consolidation in the residual left lower lobe suggesting primary bronchogenic carcinoma with central necrosis. At operation completion pneumonectomy was performed. Macroscopically, in both cases multiple and sometimes confluent foci of parenchymal consolidation were present. Microscopically these lesions corresponded to areas of lipoid pneumonia: alveolar spaces filled by lipid-laden macrophages together with slight interstitial lymphomononuclear infiltrate and prominent lymphoid follicles were observed.

Exogenous lipoid pneumonia occurs in the majority of cases in elderly patients as a complication of an underlying disease, namely, central nervous system disorders, impaired general conditions, or feeding or swallowing disturbancies. It has also been described as an occupational hazard for fire eaters or high-altitude flyers who routinely use petroleum jelly around the mask. A number of pulmonary patterns have been described; most of these patterns display clinical and roentgenological similarity with lung cancer. The diagnosis of lipoid pneumonia may be achieved noninvasively: the computed tomographic appearance seems to be characteristic [4] and bronchoalveolar lavage and fine-needle aspiration allow the clinician to achieve the correct diagnosis in the majority of cases [5]. Unfortunately, in both of our patients preoperative work-up proved negative for lipoid pneumonia and operation was deemed necessary to achieve diagnosis and for cure of possible malignancy.

In conclusion, we believe that only evidence of lipoid pneumonia proved by tissue diagnosis should be accepted in patients with known laryngeal carcinoma, and that invasive procedures, including thoracotomy, are indicated to achieve the correct diagnosis. The awareness that lipoid pneumonia may be caused in laryngectomy patients by excessive inhalation of oil drops and the similarity of this condition to malignant lesions is important in the follow-up of this increasingly large group of patients.

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References

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2. Pinkerton H. Oils and fats: their entrance into and fate in the lungs of infants and children; a clinical and pathologic report. Am J Dis Child 1927;33:259.
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Malignant Tracheobronchial Obstruction

To the Editor:

We read with interest Dr McElvein's editorial [1] on the treatment of malignant tracheobronchial obstruction, and concur with much of what he says, in particular his reference to the need for highly skilled anesthesia employing a pulse oximeter. He mentions the numerous modifications of the original Montgomery tube and refers to a bifurcated silicone rubber stent that sits in both the trachea and main bronchi.

In the last year we have been using stents, both bifurcated and straight, that can be inserted entirely endoscopically, without the need to perform a tracheostomy. We have treated 5 patients with tracheal obstruction arising as a result of malignant disease (unpublished results). Two patients had squamous carcinoma of the trachea, which was due in 1 case to radionecrosis of the tracheal cartilages 4 months after radical radiotherapy. Both of these patients were stented and derived useful palliation from the procedure. One patient had a tracheoesophageal fistula 2 years after esophagogastrectomy. This was managed by synchronous insertion of an Atkinson tube and a bifurcated tracheal stent. Unfortunately the patient died five days after the procedure from a massive hemoptysis. Of the remaining 2 patients 1 had local recurrence 2 years after a right upper lobectomy for adenocarcinoma of the bronchus and the other had stridor due to mediastinal lymph node enlargement. The former was stented and derived good palliation from the procedure. The latter, in whom the tissue diagnosis was not known, was stented and underwent mediastinoscopy at the same time. She received urgent radiotherapy the following day, the histology of the lesion was found to be small cell carcinoma, and she died from metastatic disease 14 days later.

This heterogeneous group illustrates the role that these tubes may have to play. Four of the 5 patients that we have treated had stridor arising as a result of extrinsic compression; this would not have been amenable to either laser treatment or "coring out" with a rigid bronchoscope. The stents provide instant relief of stridor, and we have not had any problems with tubes migrating. All our patients have been able to cough adequately and clear distal secretions. We believe that these stents have no serious disadvantage when compared with those that require the formation of a tracheostomy, and are considerably easier to insert.

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Reference

1. McElvein RB. Treatment of malignant tracheobronchial obstruction. Ann Thorac Surg 1989;48:463–4.

Reply

To the Editor:

Dr Gaer and associates report their use of airway stents for malignant tracheobronchial obstruction. The original paper by Mathison and Grillo [1] described their method in dealing with exophytic disease. My editorial [2] addressed the group of patients they described.

I agree with Gaer and associates that airway stents are applicable when there is extrinsic compression or destruction of the airway not amenable to local ablative techniques. Gaer and associates reinforce the idea that patients with malignant obstruction of the airway are not hopeless but can be treated with a variety of methods to achieve palliation. We have also used stents without a tracheostomy and will continue to do so.

The comments and experience of Gaer and associates add to the sparse literature about the use of stents in these circumstances.

Richard B. McElvein, MD

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1. Mathisen DJ, Grillo HC. Endoscopic relief of malignant airway obstruction. Ann Thorac Surg 1989;48:469–75.
2. McElvein RB. Treatment of malignant tracheobronchial obstruction. Ann Thorac Surg 1989;48:463–4.

PRELIMINARY PROGRAM

1990 INTERIM MEETING
THE SOCIETY OF THORACIC SURGEONS

September 21–23, 1990
Hyatt Regency Chicago, Chicago, Illinois

Friday, September 21, 1990

1:00 **Welcoming Remarks**
W. Gerald Rainer, President

Introduction to Program
Richard P. Anderson, Secretary and Interim Meeting Program Chairman

1:10–2:45 **Medico-Legal Session**
Moderator: Thomas D. Bartley, Chairman, Committee on Medico-Legal Affairs

STS Professional Liability Survey
Thomas D. Bartley

The AMA/Specialty Society Medical Liability Project
James S. Todd
Acting Executive Vice President, American Medical Association

California Tort Reform
Winfield J. Wells
Member, Committee on Medico-Legal Affairs

A Trial Lawyer's Perspective
Joseph D. Jamail
From the firm of Jamail and Kolius, Houston, TX

INTERMISSION

3:30–5:00 **Health Care in America**
Moderator: Richard P. Anderson

Health Care Needs—The Consumer Viewpoint
Robert E. McGarrah, Jr
Director for Public Policy, American Federation of State, County, and Municipal Employees

Health Care—A Political Perspective
The Honorable Richard D. Lamm
Professor, Leo Block Chair, Director, Center for Public Policy and Contemporary Issues, The University of Denver; Governor of Colorado, 1975–1987

The Health Care Industry
Austin Ross
Vice President and Executive Administrator, Virginia Mason Medical Center; Clinical Professor, University of Washington School of Public Health and Community Medicine; Chairman, Ad Hoc Committee on Physician Involvement, American Hospital Association; Consultant, W.K. Kellogg Foundation

Saturday, September 22, 1990

7:00–11:00 Two workshops will address reimbursement and coding.

7:00–8:30 **Physician's Breakfast Session**
This session will feature a representative from HCFA discussing recent changes affecting cardiothoracic reimbursement.

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9:00–11:30 **Coding Workshop for Office Personnel** (Physicians welcome)
Experts on CPT-4 and ICD-9 will explain the two coding systems and answer questions related to cardiothoracic billing.

8:30–9:45 **Plenary Session**
Moderator: W. Gerald Rainer

The Canadian Experience
Hugh E. Scully
Member, Committee on Physician Reimbursement

Thoracic Surgery Initiatives
George E. Miller, Jr
Chairman, Committee on Physician Reimbursement

INTERMISSION

10:30–12:00 **Plenary Session**

Paul B. Ginsburg
Executive Director, Physician Payment Review Commission
Washington, DC

Monica Noether
Senior Economist, Abt Associates, Inc
Cambridge, Massachusetts

Glenn R. Markus
STS Consultant on Physician Reimbursement and Government Relations
Health Policy Alternatives, Inc
Washington, DC

12:00–1:30 Luncheon: Invited Speaker
The Honorable Louis W. Sullivan
Secretary, Department of Health and Human Services

AFTERNOON SCIENTIFIC PROGRAM

2:00–5:45 **THREE PARALLEL SCIENTIFIC SESSIONS**

General Thoracic Surgery

2:00 Management of Stage IIIA Lung Cancer
Robert J. Ginsberg, New York, NY

2:20 Management of Barrett's Esophagus
F. Henry Ellis, Jr, Burlington, MA

2:40 Esophageal Diverticula
Mark B. Orringer, Ann Arbor, MI

3:00 Pulmonary Resection for Metastatic Disease
Thomas R. J. Todd, Toronto, Ont, Canada

INTERMISSION

4:10 Multimodality Treatment of Cancer of the Esophagus
Joseph I. Miller, Atlanta, GA

4:30 Mesothelioma
Nael Martini, New York, NY

Parallel Scientific Sessions, continued

4:50 Pancoast Tumor
Harold C. Urschel, Jr, Dallas, TX

5:10 Management of Achalasia
Mark K. Ferguson, Chicago, IL

Adult Cardiac Surgery

2:00 Challenges for Cardiac Transplantation in the Nineties
Bartley P. Griffith, Pittsburgh, PA

2:20 Heart-Lung Transplantation—1990
Bruce A. Reitz, Baltimore, MD

2:40 Current Status of Mechanical Circulatory Support
D. Glenn Pennington, St. Louis, MO

3:00 Cardiac Arrhythmia Surgery: Current Status and Future Directions
James L. Cox, St. Louis, MO

INTERMISSION

4:10 Surgery for Congenital Heart Disease in Adults
James K. Kirklin, Birmingham, AL

Valve Selection for Valve Replacement

4:30 Mechanical Valves
Cary W. Akins, Boston, MA

4:50 Bioprosthetic Valves
William R. E. Jamieson, Vancouver, BC, Canada

5:10 Homograft Valves
Robert B. Karp, Chicago, IL

5:30–5:45 Panel Discussion: Questions and Answers

Pediatric Cardiac Surgery

2:00 Fontan Procedure: Techniques and Results With Tricuspid Atresia and Complex Single Ventricle
Gordon K. Danielson, Rochester, MN

2:20 The Arterial Switch Operation for Simple and Complex Forms of Transposition
Edward L. Bove, Ann Arbor, MI

2:40 Surgical Management and Results in Infants with Pulmonary Atresia With Intact Ventricular Septum
William G. Williams, Toronto, Ont, Canada

3:00 Surgical Management of Coarctation in Infants and Children
Ralph D. Siewers, Pittsburgh, PA

INTERMISSION

4:10 Aortic, Mitral, and Pulmonary Valve Repair in Infants and Children
Aldo R. Castaneda, Boston, MA

4:30 Repair of Truncus Arteriosus in Infants
Kevin Turley, San Francisco, CA

4:50 Surgical Management of Tetralogy of Fallot
Vaughn A. Starnes, Stanford, CA

5:10 Anomalous Origin of the Left Coronary Artery From the Pulmonary Artery
John W. Brown, Indianapolis, IN

Sunday, September 23, 1990

7:00–10:30 **Two sessions on STS National Database**

7:00–8:00 **Breakfast Session for Members and Guests**
Presentation and discussion of collated data on adult cardiac and lung cancer surgery from STS National Database

8:30–10:30 **Workshop**
Detailed presentations, hands on demonstration, and teaching of use of STS National Database designed for surgeons, clinical research coordinators, physician assistants, and office personnel of current and prospective users.

8:00–10:00 **PLENARY SESSION**

8:00 **Lung Transplantation**
Joel D. Cooper, St. Louis, MO

8:40 **Hypoplastic Left Heart Syndrome**
Moderator: Aldo R. Castaneda, Boston, MA

Surgical Repair
William I. Norwood, Jr, Philadelphia, PA

Cardiac Transplantation
Constantine Mavroudis, Chicago, IL

9:20 **Coronary Revascularization**

Angioplasty—Current Indications and Results
Spencer B. King, Atlanta, GA

Coronary Bypass—Current Indications and Results
Floyd D. Loop, Cleveland, OH

10:45–12:00 **THREE PARALLEL HOW-TO-DO-IT SESSIONS**

General Thoracic Surgery

10:45 Management of Infected Median Sternotomy Wounds
Geoffrey M. Graeber, Morgantown, WV

11:00 Intrathoracic Lung Cancer Staging
G. Alexander Patterson, Toronto, Ont, Canada

11:15 Sleeve Lobectomy
L. Penfield Faber, Chicago, IL

11:30 Ivor-Lewis Esophagogastrectomy
Victor F. Trastek, Rochester, MN

11:45 Postpneumonectomy Empyema
Peter C. Pairolero, Rochester, MN

Adult Cardiac Surgery

10:45 Revascularization With the Gastroepiploic Artery
Bruce W. Lytle, Cleveland, OH

11:00 Retrograde Cardioplegia
W. Randolph Chitwood, Jr, Greenville, NC

11:15 Adult Homograft Aortic Valve Replacement: Surgical Challenges Requiring a Spectrum of Techniques for Solution
Richard A. Hopkins, Washington, DC

11:30 Mitral Valve Repair—1990
Delos M. Cosgrove III, Cleveland, OH

11:45 Treatment of Aortic Dissection—Updated
E. Stanley Crawford, Houston, TX

Parallel How-to-Do-It Sessions, continued

Pediatric Cardiac Surgery

10:45 Postoperative Management of Pulmonary Hypertension in Infants and Children Including ECMO
James A. Alexander, Gainesville, FL

11:00 Atrioventricular Septal Defect Repair in Infancy
Frank M. Midgley, Washington, DC

11:15 What to Do If It Can't Be Fixed: Closed Palliative Procedures
Anthony L. Moulton, Omaha, NE

11:30 Surgical Ablation of Arrhythmias in Children
David A. Ott, Houston, TX

11:45 Cryopreserved Allograft Valve Conduits and Vascular Tissue in the Repair of Complex Congenital Heart Disease
David R. Clarke, Denver, CO

12:00 ADJOURNMENT

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KEY TO ABBREVIATIONS USED FOR ARTICLE CLASSIFICATION

* = Original Article
AN = Announcement
CL = Classics in Thoracic Surgery
CR = Case Report

CP = Correspondence
CT = Collective Review
CV = Current Review
DI = Discussion
ED = Editorial
HW = How to Do It

KR = Key References
PA = Presidential Address
RE = Report
RV = Review of Recent Books

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* = Original Article
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CL = Classics in Thoracic Surgery
CR = Case Report
CP = Correspondence
CT = Collective Review
CV = Current Review
ED = Editorial
HW = How to Do It
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Incidence and risk of reintervention after coarctation repair (Kron, Flanagan, & others) 1990;49:920–6*
Isolated myocardial perfusion during arch repair (Sano & Mee) 1990;49:970–2*
Long-term results of direct aortoplasty for repair of aortic coarctation in adults (Heikkinen & Ala-Kulju) 1990;49:948–50*
Unusually early recoarctation after extended end-to-end anastomosis in an neonate (Maehara, de Leval, & Elliott) 1990;49:814–5,CR
Aortic rupture
Ascending aortic dissection in weight lifters with cystic medial degeneration (de Virgilio, Nelson, & others) 1990;49:638–42*
Thoracic aortic aneurysms after acute type A aortic dissection: necessity for follow-up (Heinemann, Laas, & others) 1990;49:580–4*
Aortic valve
Aortic homograft for aortic valve endocarditis (Wood) 1990;49:342,CP
Aortic valve debridement by ultrasonic surgical aspirator: a word of caution (Craver) 1990;49:746–53*
Aortic valve decalcification: history repeated with a new result (Cosgrove, Ratliff, & others) 1990;49:689–90,ED
Aortic valve disruption after percutaneous aortic balloon valvoplasty (Brdlik, Laub, & others) 1990;49:822–3,CR
Aortic valve operation after percutaneous aortic balloon valvuloplasty (Johnson, Dhillon, & others) 1990;49:740–5*
Aortic valvoplasty for traumatic aortic insufficiency: a 2-year follow-up (Ovil, Wahi, & others) 1990;49:143–4,CR
Early manifestation of noncalcific aortic stenosis after porcine valve replacement (Amidi, Ferson, & others) 1990;49:471–2,CR
Effect of storage at 4°C in a nutrient medium on antigenic properties of rat aortic valve allografts (El Khatib, Thompson, & others) 1990;49:792–6*
Results of homograft aortic valve replacement for active endocarditis (Tuna, Orszulak, & others) 1990;49:619–24*
Retrograde coronary sinus cardioplegia for aortic valve operations: a clinical report on 500 patients (Menasche, Subayi, & Piwnica) 1990;49:556–64*
Survival after embolization of a complete prosthetic aortic valve to the aortic arch (Stoneburner, Tucker, & Hurvitz) 1990;49:140–2,CR
Ten-year experience with aortic valve replacement in 482 patients 70 years of age or older: operative risk and long-term results (Galloway, Colvin, & others) 1990;49:84–93
Transatrial relief of diffuse subaortic stenosis after ventricular septal defect closure (DeLeon, Ilbawi, & others) 1990;49:429–34*
Use of aortic valve allografts from hepatitis B surface antigen–positive donors (Morris, Strickett, & Barratt-Boyes) 1990;49:802–5*
Ventricular septal defect associated with aortic valve incompetence: results of two surgical managements (Chauvaud, Serraf, & others) 1990;49:875–80*
Aortocoronary bypass. *See* Coronary artery bypass.
Aortopulmonary septal defect
One-stage repair of absence of the aortopulmonary septum and interrupted aortic arch (Ding, Su, & others) 1990;49:664–6,CR
Aprotinin
Aprotinin and open heart operations (Frater) 1990;49:851,CP
Arrhythmias
Computerized activation sequence mapping of the human atrial septum (Chang, Schuessler, & others) 1990;49:231–41*
Evolving patterns in the surgical treatment of malignant ventricular tachyarrhythmias (Elefteriades, Biblo, & others) 1990;49:94–100*
Surgery for ventricular arrhythmia (Moran) 1990;49:837–9,CL
Arteries
Effect of omental, intercostal, and internal mammary artery pedicle wraps on bronchial healing (Turrentine, Kesler, & others) 1990;49:574–9*
Assisted circulation
Effect of circulatory assist devices on stunned myocardium (Bavaria, Furukawa, & others) 1990;49:123–8*
Experience with univentricular support in mortally ill cardiac transplant candidates (Kormos, Borovetz, & others) 1990;49:261–72*
First human use of the Hemopump, a catheter-mounted ventricular assist device (Frazier, Wampler, & others) 1990;49:299–304*
Implantable rate-responsive counterpulsation assist system (Li, Hill, & others) 1990;49:356–62*
Atherosclerosis
Successful coronary artery bypass grafting for high-risk left main coronary artery atherosclerosis after cardiac transplantation (Copeland, Butman, & Sethi) 1990;49:106–10*
Atrium. *See* Heart atrium.
Automatic implantable cardioverter defibrillator
Evolving patterns in the surgical treatment of malignant ventricular tachyarrhythmias (Elefteriades, Biblo, & others) 1990;49:94–100*

Bacterial infections
Bacteremia and sternal infection after coronary artery bypass grafting (Kohman, Coleman, & Parker) 1990;49:454–7*
Balloon dilation
Aortic valve disruption after percutaneous aortic balloon valvoplasty (Brdlik, Laub, & others) 1990;49:822–3,CR
Aortic valve operation after percutaneous aortic balloon valvoplasty (Johnson, Dhillon, & others) 1990;49:740–5*

Fatal left ventricular perforation during mitral balloon valvoplasty (Robertson, de Virgilio, & others) 1990;49:819–21,CR
Management of neonatal critical pulmonic stenosis in the balloon valvotomy era (Caspi, Coles, & others) 1990;49: 273–8*
Operative findings after percutaneous mitral dilation (Acar, Deloche, & others) 1990;49:959–63*

Barrett's esophagus
Controversies about Barrett's esophagus (Skinner) 1990;49: 523–4,ED
Effect of antireflux operation on Barrett's mucosa (Williamson, Ellis, & others) 1990;49:537–42*
Indications for esophagectomy in nonmalignant Barrett's esophagus: a 10-year experience (Altorki, Skinner, & others) 1990;49:724–7*

Biomechanics
Experience with univentricular support in mortally ill cardiac transplant candidates (Kormos, Borovetz, & others) 1990; 49:261–72*

Bioprosthesis
Ten-year experience with porcine bioprosthetic valve: interrelationship of valve survival and patient survival in 1,050 valve replacements (Jones, Weintraub, & others) 1990; 49:370–84*

Biopsy
Alternate technique for biopsy in lung transplantation patients (Madoff) 1990;49:342–3,CP
Retrospective analysis of the efficacy of open lung biopsy in acquired immunodeficiency syndrome (Bonfils-Roberts, Nickodem, & Nealon) 1990;49:115–7*

Björk-Shiley valve
Björk-Shiley strut fracture and disc escape (Lochridge) 1990; 49:166,CP

Blalock-Taussing shunts
Modified Blalock-Taussig shunts: results in infants less than 3 months of age (Tamisier, Vouhe, & others) 1990;49:797–801*

Blood cells
Platelet and neutrophil activation in cardiopulmonary bypass (Colman) 1990;49:32–4*

Blood circulation
Assessment of spinal cord blood supply during aortic operations (Blaisdell) 1990;49:522,ED
Can AIDS be prevented after injury with contaminated instruments? (Robicsek, Duncan, & others) 1990;49:984–6*
Controlled initial hyperkalemic reperfusion after cardiac transplantation: coronary vascular resistance and blood flow (Kirklin, Neves, & others) 1990;49:625–31*
Preliminary report of localization of spinal cord blood supply by hydrogen during aortic operations (Svensson, Patel, & others) 1990;49:528–36*

Blood platelets
Intraoperative management of patients with heparin-induced thrombocytopenia (Kappa, Fisher, & others) 1990;49:714–23*

Blood pressure
Ascending aortic dissection in weight lifters with cystic medial degeneration (de Virgilio, Nelson, & others) 1990;49:638–42*
Distal aortic pressure during coarctation operation (Watterson, Dhasmana, & others) 1990;49:987–90*
Partial biventricular repair for double-outlet right ventricle with left ventricular hypoplasia (Day, Laks, & others) 1990;49:1003–5,CR

Blood transfusion
Comparative study of Cell Saver and ultrafiltration nontransfusion in cardiac surgery (Nakamura, Masuda, & others) 1990;49:973–8*
Effects of intraoperative plasmapheresis on blood loss in cardiac surgery (Jones, McCoy, & others) 1990;49:585–90*

Blood volume
Effects of intraoperative plasmapheresis on blood loss in cardiac surgery (Jones, McCoy, & others) 1990;49:585–90*

Bone neoplasms
Survival after surgical resection for high-grade chest wall sarcomas (Perry, Venzon, & others) 1990;49:363–9*

Book reviews
Cardiac Arrhythmias, 3rd edition, by Bennett (Peters) 1990;49: 499,RV
Diagnosis of Diseases of the Chest, 3rd Edition, Volume II, by Fraser, Pare, & others (Moores) 1990;49:954,RV
Heart and Heart-Lung Transplantation, edited by Wallwork (Miller) 1990;49:805,RV
Manual of Perioperative Care in Cardiac and Thoracic Surgery, by Bojar (Kriett) 1990;49:663,RV
New Trends in Heart Transplantation (Bibliotheca Cardiologica, No. 43. Series Editors, J.J.Kellermann and Tel Hasomer) edited by Wolner, Laczkovics, & Havel (Mullany) 1990;49:658,RV
Pediatric Brain Death and Organ/Tissue Retrieval. Medical, Ethical, and Legal Aspects, edited by Kaufman (Kaye) 1990;49: 774,RV
Thymic Tumors. 7th Cancer Research Workshop, Grenoble, October 1987, edited by Sarrazin, Vrousos, & Vincent (Shennib) 1990;49:151,RV

Brachytherapy
Role of brachytherapy in the management of pulmonary and mediastinal malignancies (Lewis, Ajlouni, & others) 1990; 49:728–33*

Bromocriptine
Bromocriptine as an adjuvant to cyclosporine immunosuppression after heart transplantation (Carrier, Wild, & others) 1990;49:129–32*

Bronchi
Congenital bronchoesophageal fistula (Risher, Arensman, & Ochsner) 1990;49:500–5,CT
Effect of omental, intercostal, and internal mammary artery pedicle wraps on bronchial healing (Turrentine, Kesler, & others) 1990;49:574–9*
Malignant tracheobronchial obstruction (Gaer, Townsend, & Fountain) 1990;49:1027,CP; Reply (McElvein) 1990;49: 1027,CP
Successful surgical management of bronchial dehiscence after single-lung transplantation (Kirk, Conacher, & others) 1990;49:147–9,CR
Surgery of giant bulla with tube drainage and bronchofiberoptic bronchial occlusion (Oizumi, Hoshi, & others) 1990;49:824–5,CR
Use of Gianturco self-expandable stents in the tracheobronchial tree (Varela, Maynar, & others) 1990;49:806–9*

Bronchial artery
Direct revascularization of bronchial arteries for lung transplantation: an anatomical study (Schreinemakers, Weder, & others) 1990;49:44–54*

Bronchiolitis
Improved technique for bilateral lung transplantation: rationale and initial clinical experience (Pasque, Cooper, & others) 1990;49:785–91*
Potential role of dendritic cells in bronchiolitis obliterans in heart-lung transplantation (Yousem, Ray, & others) 1990; 49:424–8*

Bulla
  Surgery of giant bulla with tube drainage and bronchofiberoptic bronchial occlusion (Oizumi, Hoshi, & others) 1990;49:824–5,CR

Calcification
  Repair of the calcified ventricular aneurysm (Cooley) 1990;49:489–90,HW
Calcium
  Postischemic [Ca^{2+}] repletion improves cardiac performance without altering oxygen demands (Yokoyama, Julian, & others) 1990;49:894–902*
Calcium channel blocker
  Endothelial cell thromboxane production and its inhibition by a calcium channel blocker (Ramadan, Upchurch, & others) 1990;49:916–9*
Candidiasis
  Candida mediastinitis after a cardiac operation (Glower, Douglas, & others) 1990;49:157–63,CV
  Disruption of the aortic anastomosis after heart-lung transplantation (Dowling, Baladi, & others) 1990;49:118–22*
Carcinoma, esophageal
  Effect of antireflux operation on Barrett's mucosa (Williamson, Ellis, & others) 1990;49:537–42*
  Surgery for high cervical esophageal carcinoma: experience with 11 patients (Mansour, Picone, & Coleman) 1990;49:597–602*
  Esophageal carcinoma in young patients (Mori, Ohno, & others) 1990;49:284–6*
Carcinoma, lung
  Predicted pulmonary function and survival after pneumonectomy for primary lung carcinoma (Putnam, Lammermeier, & others) 1990;49:909–15*
Carcinoma, non-small cell lung
  Cancer recurrence after resection: T1 N0 non-small cell lung cancer (Thomas, Rubinstein, & others) 1990;49:242–7*
  Survival after conservative resection for T1 N0 M0 non-small cell lung cancer (Read, Yoder, & Schaeffer) 1990;49:391–400*
Carcinoma, small cell
  Operation for N2 small cell lung carcinoma (Salzer, Muller, & others) 1990;49:759–62*
  Operation for small cell lung cancer—where are we? (Ginsberg) 1990;49:692–3,ED
  Operation for stage IIIa small cell lung cancer? (Bunn) 1990;49:691,ED
Cardiac massage
  The introduction of closed chest cardiac massage (Bolling) 1990;49:154–6,CL
Cardiac pacing, artificial
  Forceps extraction of permanent pacing leads (Kratz, Leman, & Gillette) 1990;49:676–7,HW
Cardiac tamponade
  Atrial pressure and hormonal and renal responses to acute cardiac tamponade (Hynynen, Salmenpera, & others) 1990;49:632–7*
Cardiomyopathy
  Operation for hypertrophic cardiomyopathy (Browdie, Agnew, & others) 1990;49:1024–5,CP; Reply (Walker) 1990;49:1025,CP
Cardioplegia. *See* Heart arrest, induced
Cardiopulmonary bypass
  Cardiac hemangioma with papillary endothelial hyperplasia: report of a resected case and review of the literature (Camp, Estruch, & others) 1990;49:305–8,CR
  Case-matched comparison of mitral valve replacement and repair (Craver, Cohen, & Weintraub) 1990;49:964–9*
  Comparative study of Cell Saver and ultrafiltration nontransfusion in cardiac surgery (Nakamura, Masuda, & others) 1990;49:973–8*
  Hypoxemia after intraluminal oxygen line obstruction during cardiopulmonary bypass (Dickinson) 1990;49:512,CP; Reply (Robblee, Crosby, & Keon) 1990;49:512,CP; Reply (Peirce) 1990;49:512–3,CP
  Improved patient survival after cardiac arrest using a cardiopulmonary support system (Reichman, Joyo, & others) 1990;49:101–5*
  Isolated myocardial perfusion during arch repair (Sano & Mee) 1990;49:970–2*
  Low activated coagulation time during cardiopulmonary bypass does not increase postoperative bleeding (Metz & Keats) 1990;49:440–4*
  Oxygenator failure (Kurusz, Conti, & Arens) 1990;49:511,CP; Reply (Robblee, Crosby, & Keon) 1990;49:512,CP; Reply (Peirce) 1990;49:512–3,CP
  Platelet and neutrophil activation in cardiopulmonary bypass (Colman) 1990;49:32–4*
  Reduction of complement activation during bypass by prime manipulation (Bonser, Dave, & others) 1990;49:279–83*
  Skeletonization of the atrioventricular node for AV node reentrant tachycardia: experience with 32 patients (Guiraudon, Klein, & others) 1990;49:565–73*
  Use of bovine lung heparin to obviate anaphylactic shock caused by porcine gut heparin (Harada, Tatsuno, & others) 1990;49:826–7,CR
Case reports
  Aortic valve disruption after percutaneous aortic balloon valvoplasty (Brdlik, Laub, & others) 1990;49:822–3,CR
  Aortic valvoplasty for traumatic aortic insufficiency: a 2-year follow-up (Ovil, Wahi, & others) 1990;49:143–4,CR
  Cardiac hemangioma with papillary endothelial hyperplasia: report of a resected case and review of the literature (Camp, Estruch, & others) 1990;49:305–8,CR
  Chiari network entrapment of thromboemboli: congenital inferior vena cava filter (Goedde, Conetta, & Rumisek) 1990;49:317–8,CR
  Congenital aneurysm of the left atrial wall in infancy (Stone, Brown, & others) 1990;49:476–8,CR
  Cor triatriatum, coarctation of the aorta, and ventricular septal defect: a rare, surgically correctable anomaly (Yamaguchi, Nishikawa, & others) 1990;49:473–5,CR
  Coronary artery bypass in a 15-year-old girl with pseudoxanthoma elasticum (Nishida, Endo, & others) 1990;49:483–5,CR
  Coronary artery bypass grafting with unanticipated fibrosing mediastinitis (Holman, Sethi, & Scott) 1990;49:321–2,CR
  Cystic pulmonary hamartoma (Miura, Hori, & others) 1990;49:828–9,CR
  Descending necrotizing mediastinitis: transcervical drainage is not enough (Wheatley, Stirling, & others) 1990;49:780–4,CR
  Dissection of the left main coronary artery: diagnosis by angioscopy (Richens, Renzulli, & Hilton) 1990;49:469–70,CR
  Dumbbell metastatic cystosarcoma phyllodes of the heart and lung (Fleisher, Tyers, & others) 1990;49:309–11,CR
  Early manifestation of noncalcific aortic stenosis after porcine valve replacement (Amidi, Ferson, & others) 1990;49:471–2,CR
  Echocardiographic diagnosis and surgical treatment of postpartum pulmonary embolism (Rosenberg, Lefor, & others) 1990;49:667–9,CR
  Esophagogastrectomy via left thoracophrenotomy (Page, Khalil, & others) 1990;49:763–6,CR

Fatal left ventricular perforation during mitral balloon valvoplasty (Robertson, de Virgilio, & others) 1990;49:819–21,CR
Free arterial graft as internal mammary artery–Y complex (Koike, Suma, & others) 1990;49:656–8,CR
Group F streptococcal empyema from aspiration of a grass inflorescence (Baethge, Eggerstedt, & Olash) 1990;49:319–20,CR
Intracavitary melanoma of the left atrium (Canver, Lajos, & others) 1990;49:312–3,CR
Management of major tracheal hemorrhage after repair of complex congenital heart defects (Snir, Carotti, & Stark) 1990;49:661–3,CR
Melanotic schwannoma of the sympathetic ganglia: pathological and clinical characteristics (Abbott, Hill, & others) 1990;49:1006–8,CR
Multiple aneurysms of the coronary arteries in a patient with systemic aneurysmal disease (LaMendola, Culliford, & others) 1990;49:1009–10,CR
Multiple congenital aneurysms of the atria (Okita, Miki, & others) 1990;49:672–3,CR
Multiple free (aorta–coronary) gastroepiploic artery grafting (Tanimoto, Matsuda, & others) 1990;49:479–80,CR
Myocardial abscess in a patient with AIDS-related complex: pericardial patch repair (Egan, Maitland, & others) 1990;49:481–2,CR
Myocardial revascularization with the lateral costal artery (Hartman, Mawulawde, & others) 1990;49:816–8,CR
Obstructing tracheal lipoma: management of a rare tumor (Chen, Braidley, & others) 1990;49:137–9,CR
One-stage repair of absence of the aortopulmonary septum and interrupted aortic arch (Ding, Su, & others) 1990;49:664–6,CR
Open fixation of flail chest after blunt trauma (Haasler) 1990;49:993–5,CR
Operative treatment of *Fusarium* fungal infection of the lung (Lupinetti, Giller, & Trigg) 1990;49:991–2,CR
Partial biventricular repair for double-outlet right ventricle with left ventricular hypoplasia (Day, Laks, & others) 1990;49:1003–5,CR
Pleural chondrosarcoma (Bailey & Head) 1990;49:996–7,CR
Recurrence and malignant transformation of endotracheal chrondroma (Salminen, Halttunen, & others) 1990;49:830–2,CR
Right atrial myxoma originating from the inferior vena cava (Bortolotti, Faggian, & others) 1990;49:1000–2,CR
Right atrial thrombosis: association with constrictive pericarditis (Katagiri, Tanabe, & others) 1990;49:145–6,CR
Sternal wire-induced persistent chest pain: a possible hypersensitivity reaction (Fine & Karwande) 1990;49:135–6,CR
Successful surgical management of bronchial dehiscence after single-lung transplantation (Kirk, Conacher, & others) 1990;49:147–9,CR
Surgery of giant bulla with tube drainage and bronchofiberoptic bronchial occlusion (Oizumi, Hoshi, & others) 1990;49:824–5,CR
Surgical ablation for idiopathic ventricular tachycardia (Blakeman & Wilber) 1990;49:314–6,CR
Surgical cure of automatic atrial tachycardia by partial left atrial isolation (Chang, Chang, & others) 1990;49:466–8,CR
Survival after embolization of a complete prosthetic aortic valve to the aortic arch (Stoneburner, Tucker, & Hurvitz) 1990;49:140–2,CR
Three-dimensional image reconstruction for planning of a complex cardiovascular procedure (Edwards, Wind, & others) 1990;49:486–8,CR
Traumatic intercostal pulmonary hernia (Forty & Wells) 1990;49:670–1,CR
Tricuspid regurgitation with postinfarction ventricular septal defect (Feng, Singh, & Moran) 1990;49:659–60,CR
Uncommon intrathoracic extrapulmonary tumor: primary hemangiopericytoma (Biagi, Gotti, & others) 1990;49:998–9,CR
Unusually early recoarctation after extended end-to-end anastomosis in an neonate (Maehara, de Leval, & Elliott) 1990;49:814–4,CR
Use of bovine lung heparin to obviate anaphylactic shock caused by porcine gut heparin (Harada, Tatsuno, & others) 1990;49:826–7,CR

Catheterization
Dissection of the left main coronary artery: diagnosis by angioscopy (Richens, Renzulli, & Hilton) 1990;49:469–70,CR
Venous cannulation for high-flow femorofemoral bypass (Smith, Getrajdman, & Hsu) 1990;49:674–5,HW
Werner Forssmann and catheterization of the heart, 1929 (Meyer) 1990;49:497–9,CL

Cefamandole
Cefamandole versus cefonicid prophylaxis in cardiovascular surgery: a prospective study (Gelfand, Simmons, & others) 1990;49:435–9*

Cefonicid
Cefamandole versus cefonicid prophylaxis in cardiovascular surgery: a prospective study (Gelfand, Simmons, & others) 1990;49:435–9*

Cerebrospinal fluid
Cerebrospinal fluid drainage and steroids provide better spinal cord protection during aortic cross-clamping than does either treatment alone (Woloszyn, Marini, & others) 1990;49:78–83*

Chest. *See* Thorax

Chest pain
Sternal wire-induced persistent chest pain: a possible hypersensitivity reaction (Fine & Karwande) 1990;49:135–6,CR

Chiari network
Chiari network entrapment of thromboemboli: congenital inferior vena cava filter (Goedde, Conetta, & Rumisek) 1990;49:317–8,CR

Child
Distal aortic pressure during coarctation operation (Watterson, Dhasmana, & others) 1990;49:987–90*
Familial achalasia in children (Monnig) 1990;49:1019–22,CV
Partial biventricular repair for double-outlet right ventricle with left ventricular hypoplasia (Day, Laks, & others) 1990;49:1003–5,CR
Role of lung decortication in symptomatic empyemas in children (Gustafson, Murray, & others) 1990;49:940–7*
Transvenous pacing in infants and children with congenital heart disease (Spotnitz) 1990;49:495–6,HW

Chondrosarcoma
Pleural chondrosarcoma (Bailey & Head) 1990;49:996–7,CR

Chrondroma
Recurrence and malignant transformation of endotracheal chrondroma (Salminen, Halttunen, & others) 1990;49:830–2,CR

Circulatory support. *See* Assisted circulation.

Classics in thoracic surgery
Individual ligation technique for lower lobe lobectomy (Faber) 1990;49:1016–8,CL
The introduction of closed chest cardiac massage (Bolling) 1990;49:154–6,CL
C. Walton Lillehei and total correction of tetralogy of Fallot (Gott) 1990;49:328–32,CL

Classics in thoracic surgery (cont'd)
- Surgery for ventricular arrhythmia (Moran) 1990;49:837–9,CL
- Toward a durable and safe aortic root reconstruction (Helseth) 1990;49:678–9,CL
- Werner Forssman and catheterization of the heart, 1929 (Meyer) 1990;49:497–9,CL

Coagulation
- Low activated coagulation time during cardiopulmonary bypass does not increase postoperative bleeding (Metz & Keats) 1990;49:440–4*

Collective review
- Congenital bronchoesophageal fistula (Risher, Arensman, & Ochsner) 1990;49:500–5,CT

Combined modality therapy
- Perioperative multimodality management of esophageal cancer: therapeutic or investigational? (Steiger) 1990;49:345–6,ED

Complement
- Reduction of complement activation during bypass by prime manipulation (Bonser, Dave, & others) 1990;49:279–83*

Computed tomography. *See* Tomography, x-ray computed.

Computer imaging
- Computerized activation sequence mapping of the human atrial septum (Chang, Schuessler, & others) 1990;49:231–41*
- Computerized potential distribution mapping: a new intraoperative mapping technique for ventricular tachycardia surgery (Harada, Tweddell, & others) 1990;49:649–55*
- Three-dimensional image reconstruction for planning of a complex cardiovascular procedure (Edwards, Wind, & others) 1990;49:486–8,CR

Congenital defects. *See* Abnormalities.

Cor triatriatum
- Cor triatriatum, coarctation of the aorta, and ventricular septal defect: a rare, surgically correctable anomaly (Yamaguchi, Nishikawa, & others) 1990;49:473–5,CR

Coronary artery bypass
- Bacteremia and sternal infection after coronary artery bypass grafting (Kohman, Coleman, & Parker) 1990;49:454–7*
- Coronary artery bypass in a 15-year-old girl with pseudoxanthoma elasticum (Nishida, Endo, & others) 1990;49:483–5,CR
- Coronary artery bypass grafting with unanticipated fibrosing mediastinitis (Holman, Sethi, & Scott) 1990;49:321–2,CR
- Crossovers in coronary artery bypass grafting trials (Weinstein & Levin) 1990;49:847,CP; Reply (Fisher, Kaiser, & others) 1990;49:848,CP
- Effects of intraoperative plasmapheresis on blood loss in cardiac surgery (Jones, McCoy, & others) 1990;49:585–90*
- Importance of precordial electrocardiogram monitoring during coronary artery bypass grafting (Bahadir & Diethrich) 1990;49:169–70,CP
- Infusion of nifedipine after coronary artery bypass grafting decreases the incidence of early postoperative myocardial ischemia (Seitelberger, Zwolfer, & others) 1990;49:61–8*
- Reduction of myocardial injury with verapamil before aortic cross-clamping (Weinstein, Rao, & Tyras) 1990;49:419–23*
- Retrograde coronary sinus cardioplegia in redo aortocoronary bypass (Calhoun, Johnston, & others) 1990;49:687,CP; Reply (Snyder) 1990;49:688,CP
- Selective approach to sternal closure after exploration for hemorrhage following coronary artery bypass (Johnson, Gundersen, & others) 1990;49:771–4*
- Sternal wound complications after isolated coronary artery bypass grafting: early and late mortality, morbidity, and cost of care (Loop, Lytle, & others) 1990;49:179–87*
- Successful coronary artery bypass grafting for high-risk left main coronary artery atherosclerosis after cardiac transplantation (Copeland, Butman, & Sethi) 1990;49:106-10*
- True emergency coronary artery bypass surgery (Edwards, Bellamy, & others) 1990;49:603–11*

Coronary artery disease
- Extended use of bilateral internal mammary arteries for coronary artery disease (Slater, Gott, & Gray) 1990;49:1014–5,HW

Coronary disease
- Multiple free (aorta–coronary) gastroepiploic artery grafting (Tanimoto, Matsuda, & others) 1990;49:479–80,CR
- Myocardial energetics and the postischemic heart (Burkhoff) 1990;49:525–7,ED

Coronary vessels
- Circumflex exposure using a cardiac sling (Galvin & Newman) 1990;49:833–4,HW
- Dissection of the left main coronary artery: diagnosis by angioscopy (Richens, Renzulli, & Hilton) 1990;49:469–70,CR
- Multiple aneurysms of the coronary arteries in a patient with systemic aneurysmal disease (LaMendola, Culliford, & others) 1990;49:1009–10,CR

Correction, 1990;49:854

Correspondence
- Alternate technique for biopsy in lung transplantation patients (Madoff) 1990;49:342–3,CP
- Aortic homograft for aortic valve endocarditis (Wood) 1990;49:342,CP
- Aprotinin and open heart operations (Frater) 1990;49:851,CP
- Björk-Shiley strut fracture and disc escape (Lochridge) 1990;49:166,CP
- Cardiac function with glucose-insulin-potassium solution (Moulton & Robinson) 1990;49:1025–6,CP; Reply (Taegtmeyer) 1990;49:1026,CP
- The Cardiac Surgery Program of the National Heart, Lung, and Blood Institute (Lenfant) 1990;49:1023–4,CP
- Cell viability in fresh, refrigerated, and cryopreserved human heart valve leaflets (Brockbank) 1990;49:848–9,CP; Reply (Shumway) 1990;49:849,CP
- Crossovers in coronary artery bypass grafting trials (Weinstein & Levin) 1990;49:847,CP; Reply (Fisher, Kaiser, & others) 1990;49:848,CP
- Cryoanalgesia for postthoracotomy pain (Berrisford & Sabanathan) 1990;49:509,CP; Reply (Katz) 1990;49:509–10,CP
- Direct access to the paravertebral space at thoracotomy (Berrisford & Sabanathan) 1990;49:855,CP
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  - Surgical treatment of cardiac tumors: a 25-year experience (Murphy, Sweeney, & others) 1990;49:612–8*
  - Technical and scientific advances in cardiac surgery over the past 25 years (Kirklin) 1990;49:26–31
  - Technique for visualization of the functioning mitral valve (Marshall) 1990;49:1013,HW

- **Heart transplantation**
  - Bromocriptine as an adjuvant to cyclosporine immunosuppression after heart transplantation (Carrier, Wild, & others) 1990;49:129–32*
  - Controlled initial hyperkalemic reperfusion after cardiac transplantation: coronary vascular resistance and blood flow (Kirklin, Neves, & others) 1990;49:625–31*
  - Experience with univentricular support in mortally ill cardiac transplant candidates (Kormos, Borovetz, & others) 1990;49:261–72*
  - Getting the most mileage from donated hearts (Terasaki) 1990;49:177–8,ED
  - HLA histocompatibility affects cardiac transplant rejection and may provide one basis for organ allocation (DiSesa, Kuo, & others) 1990;49:220–4*
  - Prostaglandin E_2 inhibits in vitro and in vivo lymphocyte responses in allogeneic transplantation (Stone, Rosengard, & others) 1990;49:927–31*
  - Rejection and infection after pediatric cardiac transplantation (Braunlin, Canter, & others) 1990;49:385–90*
  - Successful coronary artery bypass grafting for high-risk left main coronary artery atherosclerosis after cardiac transplantation (Copeland, Butman, & Sethi) 1990;49:106–10*
- **Heart valves**
  - Aortic valve disruption after percutaneous aortic balloon valvoplasty (Brdlik, Laub, & others) 1990;49:822–3,CR
  - Cell viability in fresh, refrigerated, and cryopreserved human heart valve leaflets (Brockbank) 1990;49:848,CP; Reply (Shumway) 1990;49:848,CP
  - Determinants of survival and valve failure after mitral valve replacement (Teoh, Ivanov, & others) 1990;49:643–8*
  - Early manifestation of noncalcific aortic stenosis after porcine valve replacement (Amidi, Ferson, & others) 1990;49:471–2,CR
  - Effect of storage at 4°C in a nutrient medium on antigenic properties of rat aortic valve allografts (El Khatib, Thompson, & others) 1990;49:792–6*
  - Heart valve operations in patients with active infective endocarditis (David, Bos, & others) 1990;49:701–5*
  - Ten-year experience with aortic valve replacement in 482 patients 70 years of age or older: operative risk and long-term results (Galloway, Colvin, & others) 1990;49:84–93*
  - Use of aortic valve allografts from hepatitis B surface antigen–positive donors (Morris, Strickett, & Barratt-Boyes) 1990;49:802–5*
  - Valve repair in acute endocarditis (Dreyfus, Serraf, & others) 1990;49:706–13*
- **Heart ventricles**
  - Computerized potential distribution mapping: a new intraoperative mapping technique for ventricular tachycardia surgery (Harada, Tweddell, & others) 1990;49:649–55*
  - Dynamic aortomyoplasty to assist left ventricular failure (Chachques, Grandjean, & others) 1990;49:225–30*
  - Effect of circulatory assist devices on stunned myocardium (Bavaria, Furukawa, & others) 1990;49:123–8*
  - Effects of standard mitral valve replacement on left ventricular function (Harpole, Rankin, & others) 1990;49:866–74*
  - Evolving patterns in the surgical treatment of malignant ventricular tachyarrhythmias (Elefteriades, Biblo, & others) 1990;49:94–100*
  - Fatal left ventricular perforation during mitral balloon valvoplasty (Robertson, de Virgilio, & others) 1990;49:819–21,CR
  - Management of extensive right ventricular injury or rupture (Slater, Gott, & others) 1990;49:810–3*

Heart ventricles (cont'd)
  Partial biventricular repair for double-outlet right ventricle with left ventricular hypoplasia (Day, Laks, & others) 1990;49:1003–5,CR
  Repair of the difficult ventriculotomy (Cooley) 1990;49:150–1,HW
  Surgical ablation for idiopathic ventricular tachycardia (Blakeman & Wilber) 1990;49:314–6,CR
Heart-assist devices
  Implantable rate-responsive counterpulsation assist system (Li, Hill, & others) 1990;49:356–62*
Heart-lung transplantation
  Disruption of the aortic anastomosis after heart-lung transplantation (Dowling, Baladi, & others) 1990;49:118–22*
  Lung and heart-lung transplantation (LeGal) 1990;49:840–4,CV
  Potential role of dendritic cells in bronchiolitis obliterans in heart-lung transplantation (Yousem, Ray, & others) 1990;49:424–8*
  Surgical management of heart-lung transplantation (Reichart, Vosloo, & Holl) 1990;49:333–40,CV
Hemangioendothelioma
  Mediastinal epithelioid hemangioendothelioma (Toursarkissian, O'Connor, & Dillon) 1990;49:680–5,CV
Hemangioma
  Cardiac hemangioma with papillary endothelial hyperplasia: report of a resected case and review of the literature (Camp, Estruch, & others) 1990;49:305–8,CR
Hemangiopericytoma
  Uncommon intrathoracic extrapulmonary tumor: primary hemangiopericytoma (Biagi, Gotti, & others) 1990;49:998–9,CR
Hemoconcentration
  Comparative study of Cell Saver and ultrafiltration nontransfusion in cardiac surgery (Nakamura, Masuda, & others) 1990;49:973–8*
Hemodynamics
  Ascending aortic dissection in weight lifters with cystic medial degeneration (de Virgilio, Nelson, & others) 1990;49:638–42*
  Cerebrospinal fluid drainage and steroids provide better spinal cord protection during aortic cross-clamping than does either treatment alone (Woloszyn, Marini, & others) 1990;49:78–83*
  Dynamic aortomyoplasty to assist left ventricular failure (Chachques, Grandjean, & others) 1990;49:225–30*
  Experience with univentricular support in mortally ill cardiac transplant candidates (Kormos, Borovetz, & others) 1990;49:261–72*
  Infusion of nifedipine after coronary artery bypass grafting decreases the incidence of early postoperative myocardial ischemia (Seitelberger, Zwolfer, & others) 1990;49:61–8*
  Partial biventricular repair for double-outlet right ventricle with left ventricular hypoplasia (Day, Laks, & others) 1990;49:1003–5,CR
  Shone's anomaly: operative results and late outcome (Bolling, Iannettoni, & others) 1990;49:887–93*
Hemophilia
  Myocardial abscess in a patient with AIDS-related complex: pericardial patch repair (Egan, Maitland, & others) 1990;49:481–2,CR
Hemopump
  First human use of the Hemopump, a catheter-mounted ventricular assist device (Frazier, Wampler, & others) 1990;49:299–304*
Hemorrhage
  Hemorrhage control after removal of surgically implanted intraaortic balloon pump (Opie) 1990;49:326–7,HW
  Low activated coagulation time during cardiopulmonary bypass does not increase postoperative bleeding (Metz & Keats) 1990;49:440–4*
  Management of major tracheal hemorrhage after repair of complex congenital heart defects (Snir, Carotti, & Stark) 1990;49:661–3,CR
  Selective approach to sternal closure after exploration for hemorrhage following coronary artery bypass (Johnson, Gundersen, & others) 1990;49:771–4*
Heparin
  Intraoperative management of patients with heparin-induced thrombocytopenia (Kappa, Fisher, & others) 1990;49:714–23*
  Low activated coagulation time during cardiopulmonary bypass does not increase postoperative bleeding (Metz & Keats) 1990;49:440–4*
  Use of bovine lung heparin to obviate anaphylactic shock caused by porcine gut heparin (Harada, Tatsuno, & others) 1990;49:826–7,CR
Hepatitis
  Use of aortic valve allografts from hepatitis B surface antigen–positive donors (Morris, Strickett, & Barratt-Boyes) 1990;49:802–5*
Hernia
  Traumatic intercostal pulmonary hernia (Forty & Wells) 1990;49:670–1,CR
Histocompatibility
  HLA histocompatibility affects cardiac transplant rejection and may provide one basis for organ allocation (DiSesa, Kuo, & others) 1990;49:220–4*
HLA typing
  HLA histocompatibility affects cardiac transplant rejection and may provide one basis for organ allocation (DiSesa, Kuo, & others) 1990;49:220–4*
Honored Guest Address
  Research and the practicing surgeon (Braimbridge) 1990;49:694–700*
How to do it
  Circumflex exposure using a cardiac sling (Galvin & Newman) 1990;49:833–4,HW
  Extended use of bilateral internal mammary arteries for coronary artery disease (Slater, Gott, & Gray) 1990;49:1014–5,HW
  Forceps extraction of permanent pacing leads (Kratz, Leman, & Gillette) 1990;49:676–7,HW
  Hemorrhage control after removal of surgically implanted intraaortic balloon pump (Opie) 1990;49:326–7,HW
  Inferior epigastric artery as a conduit in myocardial revascularization: the alternative free arterial graft (Vincent, van Son, & Skotnicki) 1990;49:323–5,HW
  The Killingbeck pediatric lung retractor (Satur & Walker) 1990;49:491,HW
  Method of endoscopic esophageal intubation using a rigid esophagoscope (Gaer, Blauth, & others) 1990;49:152–3,HW
  Permanent pacemaker implantation: retropectoral position and lead insertion through the subclavian vein (Relland) 1990;49:835–6,HW
  Repair of the calcified ventricular aneurysm (Cooley) 1990;49:489–90,HW
  Repair of the difficult ventriculotomy (Cooley) 1990;49:150–1,HW
  Retrophrenic location of the internal mammary artery graft (Buxton & Knight) 1990;49:1011–2,HW
  Technique for visualization of the functioning mitral valve (Marshall) 1990;49:1013,HW

- Total anomalous pulmonary venous connection: surgical repair with a double-patch technique (Corno, Giamberti, & others) 1990;49:492–4,HW
- Transvenous pacing in infants and children with congenital heart disease (Spotnitz) 1990;49:495–6,HW
- Venous cannulation for high-flow femorofemoral bypass (Smith, Getrajdman, & Hsu) 1990;49:674–5,HW

Hydrogen
- Preliminary report of localization of spinal cord blood supply by hydrogen during aortic operations (Svensson, Patel, & others) 1990;49:528–36*

Hyperkalemia
- Controlled initial hyperkalemic reperfusion after cardiac transplantation: coronary vascular resistance and blood flow (Kirklin, Neves, & others) 1990;49:625–31*

Hypersensitivity
- Sternal wire-induced persistent chest pain: a possible hypersensitivity reaction (Fine & Karwande) 1990;49:135–6,CR
- Use of bovine lung heparin to obviate anaphylactic shock caused by porcine gut heparin (Harada, Tatsuno, & others) 1990;49:826–7,CR

Hypertension, pulmonary
- Prostaglandin E_1 and intrapulmonary shunt in cardiac surgical patients with pulmonary hypertension (Heerdt & Weiss) 1990;49:463–5*

Hypothermia, induced
- Cryoanalgesia for postthoracotomy pain (Berrisford & Sabanathan) 1990;49:509,CP; Reply (Katz) 1990;49:510,CP

Iloprost
- Intraoperative management of patients with heparin-induced thrombocytopenia (Kappa, Fisher, & others) 1990;49:714–23*

Immunosuppression
- Bromocriptine as an adjuvant to cyclosporine immunosuppression after heart transplantation (Carrier, Wild, & others) 1990;49:129–32*
- Rejection and infection after pediatric cardiac transplantation (Braunlin, Canter, & others) 1990;49:385–90*
- Surgical management of heart-lung transplantation (Reichart, Vosloo, & Holl) 1990;49:333–40,CV

Infant
- Congenital aneurysm of the left atrial wall in infancy (Stone, Brown, & others) 1990;49:476–8,CR
- Correction of total anomalous pulmonary venous connection below the diaphragm (Phillips, Kongtahworn, & others) 1990;49:734–9*
- Incidence and risk of reintervention after coarctation repair (Kron, Flanagan, & others) 1990;49:920–6*
- The Killingbeck pediatric lung retractor (Satur & Walker) 1990;49:489–90,HW
- Modified Blalock-Taussig shunts: results in infants less than 3 months of age (Tamisier, Vouhe, & others) 1990;49:797–801*
- Transvenous pacing in infants and children with congenital heart disease (Spotnitz) 1990;49:495–6,HW
- Unusually early recoarctation after extended end-to-end anastomosis in an neonate (Maehara, de Leval, & Elliott) 1990;49:814–5,CR

Infection
- Bacteremia and sternal infection after coronary artery bypass grafting (Kohman, Coleman, & Parker) 1990;49:454–7*
- *Candida* mediastinitis after a cardiac operation (Glower, Douglas, & others) 1990;49:157–63,CV
- Descending necrotizing mediastinitis: transcervical drainage is not enough (Wheatley, Stirling, & others) 1990;49:780–4,CR
- Group F streptococcal empyema from aspiration of a grass inflorescence (Baethge, Eggerstedt, & Olash) 1990;49:319–20,CR
- Life-threatening acute posterior mediastinitis due to esophageal perforation (Burnett, Rosemurgy, & Pfeiffer) 1990;49:979–83*
- Operative treatment of *Fusarium* fungal infection of the lung (Lupinetti, Giller, & Trigg) 1990;49:991–2*
- Recent experience with major sternal wound complications (Demmy, Park, & others) 1990;49:458–62*
- Rejection and infection after pediatric cardiac transplantation (Braunlin, Canter, & others) 1990;49:385–90*
- Risks of bilateral internal mammary artery bypass grafting (Kouchoukos, Wareing, & others) 1990;49:210–9*
- Sternal wound complications after isolated coronary artery bypass grafting: early and late mortality, morbidity, and cost of care (Loop, Lytle, & others) 1990;49:179–87*
- Successful surgical management of bronchial dehiscence after single-lung transplantation (Kirk, Conacher, & others) 1990;49:147–9,CR

Insulin
- Cardiac function with glucose-insulin-potassium solution (Moulton & Robinson) 1990;49:1025–6,CP; Reply (Taegtmeyer) 1990;49:1026,CP

Intensive care units
- Reoperation in the intensive care unit (Kaiser, Naunheim, & others) 1990;49:903–8*

Intestine, small
- Experimental reconstruction of the canine trachea with a free revascularized small bowel graft (Letang, Sanchez-Lloret, & others) 1990;49:955–8*

Intraaortic balloon pump
- Effect of circulatory assist devices on stunned myocardium (Bavaria, Furukawa, & others) 1990;49:123–8*
- Hemorrhage control after removal of surgically implanted intraaortic balloon pump (Opie) 1990;49:326–7,HW

Ischemia
- Infusion of nifedipine after coronary artery bypass grafting decreases the incidence of early postoperative myocardial ischemia (Seitelberger, Zwolfer, & others) 1990;49:61–8*
- Lung and heart-lung transplantation (LeGal) 1990;49:840–4,CV
- Myocardial energetics and the postischemic heart (Burkhoff) 1990;49:525–7,ED
- Relationship between total mechanical energy and oxygen consumption in the stunned myocardium (Furukawa, Bavaria, & others) 1990;49:543–9*
- Superior myocardial preservation with modified UW solution after prolonged ischemia in the rat heart (Yeh, Hanan, & others) 1990;49:932–9*
- True emergency coronary artery bypass surgery (Edwards, Bellamy, & others) 1990;49:603–11*

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- Surgical techniques for chest wall reconstruction (Mathisen) 1990;49:164–5,KR
- The trachea (Grillo & Mathisen) 1990;49:845–6,KR

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- Atrial pressure and hormonal and renal responses to acute cardiac tamponade (Hynynen, Salmenpera, & others) 1990;49:632–7*

Kouwenhoven, William
- The introduction of closed chest cardiac massage (Bolling) 1990;49:154–6,CL

Larynx
- Cervical exenteration (Grillo & Mathisen) 1990;49:401–9*
- Lipoid pneumonia after laryngectomy mimicking pulmonary malignancy (Venuta, Rendina, & others) 1990;49:1026–7,CP

Lillehei, C. Walton
- C. Walton Lillehei and total correction of tetralogy of Fallot (Gott) 1990;49:328–32,CL

Lipoma
- Obstructing tracheal lipoma: management of a rare tumor (Chen, Braidley, & others) 1990;49:137–9,CR

Lung
- Cystic pulmonary hamartoma (Miura, Hori, & others) 1990;49:828–9,CR
- Dumbbell metastatic cystosarcoma phyllodes of the heart and lung (Fleisher, Tyers, & others) 1990;49:309–11,CR
- Effect of routine fibrin glue use on the duration of air leaks after lobectomy (Fleisher, Evans, & others) 1990;49:133–4*
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- The Killingbeck pediatric lung retractor (Satur & Walker) 1990;49:489–90,HW
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- Management of neonatal critical pulmonic stenosis in the balloon valvotomy era (Caspi, Coles, & others) 1990;49:273–8*
- Operative treatment of *Fusarium* fungal infection of the lung (Lupinetti, Giller, & Trigg) 1990;49:991–2*
- Pleural chondrosarcoma (Bailey & Head) 1990;49:996–7,CR
- Retrospective analysis of the efficacy of open lung biopsy in acquired immunodeficiency syndrome (Bonfils-Roberts, Nickodem, & Nealon) 1990;49:115–7*
- Role of lung decortication in symptomatic empyemas in children (Gustafson, Murray, & others) 1990;49:940–7*
- Total anomalous pulmonary venous connection: surgical repair with a double-patch technique (Corno, Giamberti, & others) 1990;49:492–4,HW
- Traumatic intercostal pulmonary hernia (Forty & Wells) 1990;49:670–1,CR
- Uncommon intrathoracic extrapulmonary tumor: primary hemangiopericytoma (Biagi, Gotti, & others) 1990;49:998–9,CR
- Use of the argon beam electrocoagulator for performing pulmonary wedge resections (Rusch, Schmidt, & others) 1990;49:287–91*
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- Cancer recurrence after resection: T1 N0 non-small cell lung cancer (Thomas, Rubinstein, & others) 1990;49:242–7*
- Predicted pulmonary function and survival after pneumonectomy for primary lung carcinoma (Putnam, Lammermeier, & others) 1990;49:909–15*
- Operation for N2 small cell lung carcinoma (Salzer, Muller, & others) 1990;49:759–62*
- Operation for small cell lung cancer—where are we? (Ginsberg) 1990;49:692–3,ED
- Operation for stage IIIa small cell lung cancer? (Bunn) 1990;49:691,ED
- Role of brachytherapy in the management of pulmonary and mediastinal malignancies (Lewis, Ajlouni, & others) 1990;49:728–33*
- Surgery of giant bulla with tube drainage and bronchofiberoptic bronchial occlusion (Oizumi, Hoshi, & others) 1990;49:824–5,CR
- Survival after conservative resection for T1 N0 M0 non-small cell lung cancer (Read, Yoder, & Schaeffer) 1990;49:391–400*

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- Alternate technique for biopsy in lung transplantation patients (Madoff) 1990;49:342–3,CP
- Direct revascularization of bronchial arteries for lung transplantation: an anatomical study (Schreinemakers, Weder, & others) 1990;49:44–54*
- Donor-specific cytotoxicity testing: an advance in detecting pulmonary allograft rejection (Dal Col, Zeevi, & others) 1990;49:754–8*
- Improved technique for bilateral lung transplantation: rationale and initial clinical experience (Pasque, Cooper, & others) 1990;49:785–91*
- Lung and heart-lung transplantation (LeGal) 1990;49:840–4,CV
- Reduced-size lung transplantation in neonatal swine: technique and short-term physiological response (Crombleholme, Adzick, & others) 1990;49:55–60*
- Successful surgical management of bronchial dehiscence after single-lung transplantation (Kirk, Conacher, & others) 1990;49:147–9,CR

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- Prostaglandin E_2 inhibits in vitro and in vivo lymphocyte responses in allogeneic transplantation (Stone, Rosengard, & others) 1990;49:927–31*

Mammary artery
- Internal mammary artery grafts (de la Riviere, Defauw, & others) 1990;49:166,CP; Reply (Vander Salm) 1990;49:166,CP
- Risks of bilateral internal mammary artery bypass grafting (Kouchoukos, Wareing, & others) 1990;49:210–9*
- Routine use of the left internal mammary artery graft in the elderly (Gardner, Greene, & others) 1990;49:188–94*
- Sequel of the internal mammary artery as a systemic-to-pulmonary artery shunt (McKay) 1990;49:341,CP
- Seventeen-year experience with bilateral internal mammary artery grafts (Galbut, Traad, & others) 1990;49:195–201*

Mediastinitis
- *Candida* mediastinitis after a cardiac operation (Glower, Douglas, & others) 1990;49:157–63,CV
- Coronary artery bypass grafting with unanticipated fibrosing mediastinitis (Holman, Sethi, & Scott) 1990;49:321–2,CR
- Descending necrotizing mediastinitis: transcervical drainage is not enough (Wheatley, Stirling, & others) 1990;49:780–4,CR
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- Mediastinitis after cardiac operations (Spencer & Grossi) 1990;49:506–7,KR

Mediastinum
- Mediastinal epithelioid hemangioendothelioma (Toursarkissian, O'Connor, & Dillon) 1990;49:680–5,CV
- Role of brachytherapy in the management of pulmonary and mediastinal malignancies (Lewis, Ajlouni, & others) 1990;49:728–33*

Melanin
- Melanotic schwannoma of the sympathetic ganglia: pathological and clinical characteristics (Abbott, Hill, & others) 1990;49:1006–8,CR

Microscopy, electron
- Effect of storage at 4°C in a nutrient medium on antigenic properties of rat aortic valve allografts (El Khatib, Thompson, & others) 1990;49:792–6*

Mitral valve
- Case-matched comparison of mitral valve replacement and repair (Craver, Cohen, & Weintraub) 1990;49:964–9*
- Determinants of survival and valve failure after mitral valve replacement (Teoh, Ivanov, & others) 1990;49:643–8*
- Dissection of the left main coronary artery: diagnosis by angioscopy (Richens, Renzulli, & Hilton) 1990;49:469–70,CR
- Effects of standard mitral valve replacement on left ventricular function (Harpole, Rankin, & others) 1990;49:866–74*
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- Mitral valve replacement: what should be the standard technique? (Tyers) 1990;49:861–2,ED
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- Operative findings after percutaneous mitral dilation (Acar, Deloche, & others) 1990;49:959–63*
- Technique for visualization of the functioning mitral valve (Marshall) 1990;49:1013,HW

Morbidity
- True emergency coronary artery bypass surgery (Edwards, Bellamy, & others) 1990;49:603–11*

Mortality
- Esophagogastrectomy via left thoracophrenotomy (Page, Khalil, & others) 1990;49:763–6,CR
- Evolving patterns in the surgical treatment of malignant ventricular tachyarrhythmias (Elefteriades, Biblo, & others) 1990;49:94–100*
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- Results of internal thoracic artery grafting over 15 years: single versus double grafts (Fiore, Naunheim, & others) 1990; 49:202–9*
- Seventeen-year experience with bilateral internal mammary artery grafts (Galbut, Traad, & others) 1990;49:195–201*
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- Thymectomy for myasthenia gravis (Jaretzki) 1990;49:688,CP; Reply (Mulder) 1990;49:688,CP

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- Repair of the difficult ventriculotomy (Cooley) 1990;49:150–1,HW
- Tricuspid regurgitation with postinfarction ventricular septal defect (Feng, Singh, & Moran) 1990;49:659–60,CR

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- Surgical excision of intracardiac myxomas: a 20-year follow-up (Bortolotti, Maraglino, & others) 1990;49:449–53*

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- Atrial pressure and hormonal and renal responses to acute cardiac tamponade (Hynynen, Salmenpera, & others) 1990;49:632–7*

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- Descending necrotizing mediastinitis: transcervical drainage is not enough (Wheatley, Stirling, & others) 1990;49:780–4,CR

Neoplasms
- Can computed tomography of the chest stage lung cancer? Yes and no (Lewis, Pearlberg, & others) 1990;49:591–6*
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- Pleural chondrosarcoma (Bailey & Head) 1990;49:996–7,CR
- Predicted pulmonary function and survival after pneumonectomy for primary lung carcinoma (Putnam, Lammermeier, & others) 1990;49:909–15*
- Primary tracheal tumors: treatment and results (Grillo & Mathisen) 1990;49:69–77*
- Recurrence and malignant transformation of endotracheal chrondroma (Salminen, Halttunen, & others) 1990;49:830–2,CR
- Resection of atriocaval adrenal carcinoma (Theman) 1990;49:170,CP
- Role of brachytherapy in the management of pulmonary and mediastinal malignancies (Lewis, Ajlouni, & others) 1990;49:728–33*
- Surgical treatment of cardiac tumors: a 25-year experience (Murphy, Sweeney, & others) 1990;49:612–8*
- Survival after conservative resection for T1 N0 M0 non-small cell lung cancer (Read, Yoder, & Schaeffer) 1990;49:391–400*
- Uncommon intrathoracic extrapulmonary tumor: primary hemangiopericytoma (Biagi, Gotti, & others) 1990;49:998–9,CR

Neutrophils
- Platelet and neutrophil activation in cardiopulmonary bypass (Colman) 1990;49:32–4*

Nicardipine
- Clinical trial of nicardipine cardioplegia in pediatric cardiac surgery (Mori, Miyamoto, & others) 1990;49:413–8*

Nifedipine
- Infusion of nifedipine after coronary artery bypass grafting decreases the incidence of early postoperative myocardial ischemia (Seitelberger, Zwolfer, & others) 1990;49:61–8*

Organ preservation
- Superior myocardial preservation with modified UW solution after prolonged ischemia in the rat heart (Yeh, Hanan, & others) 1990;49:932–9*
- Surgical management of heart-lung transplantation (Reichart, Vosloo, & Holl) 1990;49:333–40,CV

Ossification, pathologic
- Ectopic ossification after median sternotomy (Weiman) 1990;49:510,CP

Oxygen consumption
- Open fixation of flail chest after blunt trauma (Haasler) 1990;49:993–5,CR
- Postischemic [Ca^{2+}] repletion improves cardiac performance without altering oxygen demands (Yokoyama, Julian, & others) 1990;49:894–902*
- Relationship between total mechanical energy and oxygen consumption in the stunned myocardium (Furukawa, Bavaria, & others) 1990;49:543–9*

Oxygenators
- Hypoxemia after intraluminal oxygen line obstruction during cardiopulmonary bypass (Dickinson) 1990;49:512,CP; Reply (Robblee, Crosby, & Keon) 1990;49:512,CP; Reply (Peirce) 1990;49:512–3,CP
- Oxygenator failure (Kurusz, Conti, & Arens) 1990;49:511,CP; Reply (Robblee, Crosby, & Keon) 1990;49:512,CP; Reply (Peirce) 1990;49:512–3,CP

Pacemaker, artificial
- Forceps extraction of permanent pacing leads (Kratz, Leman, & Gillette) 1990;49:676–7,HW
- Permanent pacemaker implantation: retropectoral position and lead insertion through the subclavian vein (Relland) 1990;49:835–6,HW
- Transvenous pacing in infants and children with congenital heart disease (Spotnitz) 1990;49:495–6,HW

Pain
- Cryoanalgesia for postthoracotomy pain (Berrisford & Sabanathan) 1990;49:509,CP; Reply (Katz) 1990;49:510,CP
- Sternal wire-induced persistent chest pain: a possible hypersensitivity reaction (Fine & Karwande) 1990;49:135–6,CR

Paralysis
- Diaphragmatic plication for unilateral diaphragmatic paralysis: a 10-year experience (Graham, Kaplan, & others) 1990;49:248–52*

Paraplegia
- Preliminary report of localization of spinal cord blood supply by hydrogen during aortic operations (Svensson, Patel, & others) 1990;49:528–36*

Pediatrics
- Clinical trial of nicardipine cardioplegia in pediatric cardiac surgery (Mori, Miyamoto, & others) 1990;49:413–8*
- Rejection and infection after pediatric cardiac transplantation (Braunlin, Canter, & others) 1990;49:385–90*

Pericarditis
- Futility of pericardiectomy for postirradiation constrictive pericarditis? (Ni, von Segesser, & Turina) 1990;49:445–8*
- Right atrial thrombosis: association with constrictive pericarditis (Katagiri, Tanabe, & others) 1990;49:145–6,CR

Pericardium
- Myocardial abscess in a patient with AIDS-related complex: pericardial patch repair (Egan, Maitland, & others) 1990;49:481–2,CR
- Pericardial flap (Flege) 1990;49:341,CP; Reply (Starr & Moore) 1990;49:342,CP

Plasmapheresis
- Effects of intraoperative plasmapheresis on blood loss in cardiac surgery (Jones, McCoy, & others) 1990;49:585–90*

Platelets
- Platelet and neutrophil activation in cardiopulmonary bypass (Colman) 1990;49:32–4*

Pleurodesis
- Tetracycline pleurodesis for persistent air leak (Chechani) 1990;49:166–7,CP; Reply (Almassi & Haasler) 1990;49:167,CP

Pneumonectomy
- Predicted pulmonary function and survival after pneumonectomy for primary lung carcinoma (Putnam, Lammermeier, & others) 1990;49:909–15*

Pneumonia
- Lipoid pneumonia after laryngectomy mimicking pulmonary malignancy (Venuta, Rendina, & others) 1990;49:1026–7,CP

Polygeline
- Reduction of complement activation during bypass by prime manipulation (Bonser, Dave, & others) 1990;49:279–83*

Potassium
  Cardiac function with glucose-insulin-potassium solution (Moulton & Robinson) 1990;49:1025–6,CP; Reply (Taegtmeyer) 1990;49:1026,CP
  Clinical trial of nicardipine cardioplegia in pediatric cardiac surgery (Mori, Miyamoto, & others) 1990;49:413–8*
  Controlled initial hyperkalemic reperfusion after cardiac transplantation: coronary vascular resistance and blood flow (Kirklin, Neves, & others) 1990;49:625–31*
Pregnancy
  Echocardiographic diagnosis and surgical treatment of postpartum pulmonary embolism (Rosenberg, Lefor, & others) 1990;49:667–9,CR
Presidential Address
  Philosophy 1 (Lindesmith) 1990;49:7–13,PA
  Who, Hobbies, and Heroes (Clark) 1990;49:525–21,PA
Prolactin
  Bromocriptine as an adjuvant to cyclosporine immunosuppression after heart transplantation (Carrier, Wild, & others) 1990;49:129–32*
Prophylaxis
  Cefamandole versus cefonicid prophylaxis in cardiovascular surgery: a prospective study (Gelfand, Simmons, & others) 1990;49:435–9*
Prostacyclin
  Comparison of prostacyclin production of human gastroepiploic artery and saphenous vein (Oku, Yamane, & others) 1990;49:767–70*
  Vascular properties of canine lungs perfused with Eurocollins solution and prostacyclin (Unruh, Hoppensack, & Oppenheimer) 1990;49:292–8*
Prostaglandins
  Platelet and neutrophil activation in cardiopulmonary bypass (Colman) 1990;49:32–4*
  Prostaglandin E_1 and intrapulmonary shunt in cardiac surgical patients with pulmonary hypertension (Heerdt & Weiss) 1990;49:463–5*
  Prostaglandin E_2 inhibits in vitro and in vivo lymphocyte responses in allogeneic transplantation (Stone, Rosengard, & others) 1990;49:927–31*
Prosthesis
  Case-matched comparison of mitral valve replacement and repair (Craver, Cohen, & Weintraub) 1990;49:964–9*
  Determinants of survival and valve failure after mitral valve replacement (Teoh, Ivanov, & others) 1990;49:643–8*
  Early manifestation of noncalcific aortic stenosis after porcine valve replacement (Amidi, Ferson, & others) 1990;49:471–2,CR
  Must the mitral valve always be removed during prosthetic replacement? (Campanella, Hider, & others) 1990;49:167–8,CP
  Porcine versus pericardial bioprostheses (Stassano, Gagliardi, & Spampinato) 1990;49:840,CP
  Survival after embolization of a complete prosthetic aortic valve to the aortic arch (Stoneburner, Tucker, & Hurvitz) 1990;49:140–2,CR
  Valvular xenograft to bioprosthesis (Hancock) 1990;49:170,CP
Pseudoxanthoma elasticum
  Coronary artery bypass in a 15-year-old girl with pseudoxanthoma elasticum (Nishida, Endo, & others) 1990;49:483–5,CR
Pulmonary artery
  One-stage repair of absence of the aortopulmonary septum and interrupted aortic arch (Ding, Su, & others) 1990;49:664–6,CR
  Palliative reconstruction of right ventricular outflow tract in tetralogy with hypoplastic pulmonary arteries (Okita, Miki, & others) 1990;49:775–9*
Pulmonary circulation
  Correction of total anomalous pulmonary venous connection below the diaphragm (Phillips, Kongtahworn, & others) 1990;49:734–9*
  Predicted pulmonary function and survival after pneumonectomy for primary lung carcinoma (Putnam, Lammermeier, & others) 1990;49:909–15*
Pulmonary embolism
  Chiari network entrapment of thromboemboli: congenital inferior vena cava filter (Goedde, Conetta, & Rumisek) 1990;49:317–8,CR
  Echocardiographic diagnosis and surgical treatment of postpartum pulmonary embolism (Rosenberg, Lefor, & others) 1990;49:667–9,CR
Pulmonary gas exchange
  Effect of routine fibrin glue use on the duration of air leaks after lobectomy (Fleisher, Evans, & others) 1990;49:133–4*
  Tetracycline pleurodesis for persistent air leak (Chechani) 1990;49:166–7,CP; Reply (Almassi & Haasler) 1990;49:167,CP
Pumps
  First human use of the Hemopump, a catheter-mounted ventricular assist device (Frazier, Wampler, & others) 1990;49:299–304*

Radioisotopes
  Role of brachytherapy in the management of pulmonary and mediastinal malignancies (Lewis, Ajlouni, & others) 1990;49:728–33*
Radiotherapy
  Chemotherapy and radiation therapy before transhiatal esophagectomy for esophageal carcinoma (Orringer, Forastiere, & others) 1990;49:348–55*
  Futility of pericardiectomy for postirradiation constrictive pericarditis? (Ni, von Segesser, & Turina) 1990;49:445–8*
  Mediastinal epithelioid hemangioendothelioma (Toursarkissian, O'Connor, & Dillon) 1990;49:680–5,CV
  Primary tracheal tumors: treatment and results (Grillo & Mathisen) 1990;49:69–77*
Rats
  Effect of storage at 4°C in a nutrient medium on antigenic properties of rat aortic valve allografts (El Khatib, Thompson, & others) 1990;49:792–6*
  Prostaglandin E_2 inhibits in vitro and in vivo lymphocyte responses in allogeneic transplantation (Stone, Rosengard, & others) 1990;49:927–31*
Reflux
  Effect of antireflux operation on Barrett's mucosa (Williamson, Ellis, & others) 1990;49:537–42*
Rejection
  Potential role of dendritic cells in bronchiolitis obliterans in heart-lung transplantation (Yousem, Ray, & others) 1990;49:424–8*
  Rejection and infection after pediatric cardiac transplantation (Braunlin, Canter, & others) 1990;49:385–90*
Reoperation
  Incidence and risk of reintervention after coarctation repair (Kron, Flanagan, & others) 1990;49:920–6*
  Late left atrioventricular valve insufficiency after repair of partial atrioventricular septal defects: anatomical and surgical determinants (Abbruzzese, Napoleone, & others) 1990;49:111–4*
  Reoperation in the intensive care unit (Kaiser, Naunheim, & others) 1990;49:903–8*

Reoperation (cont'd)
  Reoperation for thymoma: report of 23 cases (Kirschner) 1990;49:550–5*
  Seventeen-year experience with bilateral internal mammary artery grafts (Galbut, Traad, & others) 1990;49:195–201*
  Successful surgical management of bronchial dehiscence after single-lung transplantation (Kirk, Conacher, & others) 1990;49:147–9,CR
  Ten-year experience with porcine bioprosthetic valve: interrelationship of valve survival and patient survival in 1,050 valve replacements (Jones, Weintraub & others) 1990;49:370–84*
  Ten-year experience with aortic valve replacement in 482 patients 70 years of age or older: operative risk and long-term results (Galloway, Colvin, & others) 1990;49:84–93
  Thoracic aortic aneurysms after acute type A aortic dissection: necessity for follow-up (Heinemann, Laas, & others) 1990;49:580–4*
Report
  Notes from the Thoracic Surgery Directors Association (Murray) 1990;49:855,RE
  The use of extracorporeal circulation (ECC) for circulatory support during PTCA 1990;49:514,RE
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  Research and the practicing surgeon (Braimbridge) 1990;49:694–700*
Respiration
  Diaphragmatic plication for unilateral diaphragmatic paralysis: a 10-year experience (Graham, Kaplan, & others) 1990;49:248–52*
  Obstructing tracheal lipoma: management of a rare tumor (Chen, Braidley, & others) 1990;49:137–9,CR
  Retrospective analysis of the efficacy of open lung biopsy in acquired immunodeficiency syndrome (Bonfils-Roberts, Nickodem, & Nealon) 1990;49:115–7*
  Use of Gianturco self-expandable stents in the tracheobronchial tree (Varela, Maynar, & others) 1990;49:806–9*
Resuscitation
  The introduction of closed chest cardiac massage (Bolling) 1990;49:154–6,CL
Revascularization
  Comparison of prostacyclin production of human gastroepiploic artery and saphenous vein (Oku, Yamane, & others) 1990;49:767–70*
  Direct revascularization of bronchial arteries for lung transplantation: an anatomical study (Schreinemakers, Weder, & others) 1990;49:44–54*
  Experimental reconstruction of the canine trachea with a free revascularized small bowel graft (Letang, Sanchez-Lloret, & others) 1990;49:955–8*
  Extended use of bilateral internal mammary arteries for coronary artery disease (Slater, Gott, & Gray) 1990;49:1014–5,HW
  Inferior epigastric artery as a conduit in myocardial revascularization: the alternative free arterial graft (Vincent, van Son, & Skotnicki) 1990;49:323–5,HW
  Myocardial revascularization with the lateral costal artery (Hartman, Mawulawde, & others) 1990;49:816–8,CR
  Retrophrenic location of the internal mammary artery graft (Buxton, Knight, & others) 1990;49:1011–2,HW
Risk factors
  Hospital death on a cardiac surgical service: negative influence of changing practice patterns (McGrath, Laub, & others) 1990;49:410–2*
  Late left atrioventricular valve insufficiency after repair of partial atrioventricular septal defects: anatomical and surgical determinants (Abbruzzese, Napoleone, & others) 1990;49:111–4*
  Recent experience with major sternal wound complications (Demmy, Park, & others) 1990;49:458–62*
  Risks of bilateral internal mammary artery bypass grafting (Kouchoukos, Wareing, & others) 1990;49:210–9*
  Successful coronary artery bypass grafting for high-risk left main coronary artery atherosclerosis after cardiac transplantation (Copeland, Butman, & Sethi) 1990;49:106–10*
  True emergency coronary artery bypass surgery (Edwards, Bellamy, & others) 1990;49:603–11*

Saphenous vein
  Comparison of prostacyclin production of human gastroepiploic artery and saphenous vein (Oku, Yamane, & others) 1990;49:767–70*
Sarcoma
  Survival after surgical resection for high-grade chest wall sarcomas (Perry, Venzon, & others) 1990;49:363–9*
Schwannoma
  Melanotic schwannoma of the sympathetic ganglia: pathological and clinical characteristics (Abbott, Hill, & others) 1990;49:1006–8,CR
Secretions
  Clinical experience with minitracheostomy (Wain, Wilson, & Mathisen) 1990;49:881–6*
Sheep
  Effect of circulatory assist devices on stunned myocardium (Bavaria, Furukawa, & others) 1990;49:123–8*
  Relationship between total mechanical energy and oxygen consumption in the stunned myocardium (Furukawa, Bavaria, & others) 1990;49:543–9*
Shone's anomaly
  Shone's anomaly: operative results and late outcome (Bolling, Iannettoni, & others) 1990;49:887–93*
Shunts
  Prostaglandin E_1 and intrapulmonary shunt in cardiac surgical patients with pulmonary hypertension (Heerdt & Weiss) 1990;49:463–5*
  Sequel of the internal mammary artery as a systemic-to-pulmonary artery shunt (McKay) 1990;49:341,CP
The Society of Thoracic Surgeons
  Twenty-fifth Anniversary Meeting 1990;49:171–6
    Philosophy 1 (Lindesmith) 1990;49:7–13,PA
    Platelet and neutrophil activation in cardiopulmonary bypass (Colman) 1990;49:32–4*
    Technical and scientific advances in cardiac surgery over the past 25 years (Kirklin) 1990;49:26–31*
    Technical and scientific advances in general thoracic surgery (Skinner) 1990;49:14–25*
    Welcoming remarks (Hudson) 1990;49:4*
    Welcoming remarks (Ross) 1990;49:5*
    Welcoming remarks (Schmoke) 1990;49:6*
Spinal cord
  Assessment of spinal cord blood supply during aortic operations (Blaisdell) 1990;49:522,ED
  Cerebrospinal fluid drainage and steroids provide better spinal cord protection during aortic cross-clamping than does either treatment alone (Woloszyn, Marini, & others) 1990;49:78–83*
  Preliminary report of localization of spinal cord blood supply by hydrogen during aortic operations (Svensson, Patel, & others) 1990;49:528–36*
Sports
  Ascending aortic dissection in weight lifters with cystic medial degeneration (de Virgilio, Nelson, & others) 1990;49:638–42*
Statistics
  Ten-year experience with aortic valve replacement in 482 patients 70 years of age or older: operative risk and

long-term results (Galloway, Colvin, & others) 1990; 49:84–93

Stents

Tracheal reconstruction by esophageal interposition: an experimental study (Kato, Onuki, & others) 1990;49:951–4*

Use of Gianturco self-expandable stents in the tracheobronchial tree (Varela, Maynar, & others) 1990;49:806–9*

Sternum

Bacteremia and sternal infection after coronary artery bypass grafting (Kohman, Coleman, & Parker) 1990;49:454–7*

Ectopic ossification after median sternotomy (Weiman) 1990; 49:510,CP

Management of sternal tumors (Yellin & Lieberman) 1990;49: 852,CP; Reply (Gabbay) 1990;49:852,CP

Recent experience with major sternal wound complications (Demmy, Park, & others) 1990;49:458–62*

Repair of sternal dehiscence using a Harrington compression system (Cochran & Rohrich) 1990;49:853,CP; Reply (Johnson) 1990;49:854,CP

Selective approach to sternal closure after exploration for hemorrhage following coronary artery bypass (Johnson, Gundersen, & others) 1990;49:771–4*

Sternal wire-induced persistent chest pain: a possible hypersensitivity reaction (Fine & Karwande) 1990;49:135–6,CR

Sternal wound complications after isolated coronary artery bypass grafting: early and late mortality, morbidity, and cost of care (Loop, Lytle, & others) 1990;49:179–87*

Steroids

Cerebrospinal fluid drainage and steroids provide better spinal cord protection during aortic cross-clamping than does either treatment alone (Woloszyn, Marini, & others) 1990; 49:78–83*

Streptococcal infections

Group F streptococcal empyema from aspiration of a grass inflorescence (Baethge, Eggerstedt, & Olash) 1990;49:319–20,CR

Subclavian vein

Permanent pacemaker implantation: retropectoral position and lead insertion through the subclavian vein (Relland) 1990; 49:835–6,HW

Surgery

Cefamandole versus cefonicid prophylaxis in cardiovascular surgery: a prospective study (Gelfand, Simmons, & others) 1990;49:435–9*

Changing cardiothoracic practice patterns (Rainer) 1990; 49:347,ED

Direct revascularization of bronchial arteries for lung transplantation: an anatomical study (Schreinemakers, Weder, & others) 1990;49:44–54*

Disruption of the aortic anastomosis after heart-lung transplantation (Dowling, Baladi, & others) 1990;49:118–22*

Esophagectomy for esophageal disruption (Orringer & Stirling) 1990;49:35–43*

Primary tracheal tumors: treatment and results (Grillo & Mathisen) 1990;49:69–77*

Reduced-size lung transplantation in neonatal swine: technique and short-term physiological response (Crombleholme, Adzick, & others) 1990;49:55–60*

Surgical techniques for chest wall reconstruction (Mathisen) 1990;49:164–5,KR

Surgical treatment of automatic atrial tachycardias (Hendry, Packer, & others) 1990;49:253–60*

Survival after surgical resection for high-grade chest wall sarcomas (Perry, Venzon, & others) 1990;49:363–9*

Total anomalous pulmonary venous connection: surgical repair with a double-patch technique (Corno, Giamberti, & others) 1990;49:492–4,HW

Surgery department, hospital

Hospital death on a cardiac surgical service: negative influence of changing practice patterns (McGrath, Laub, & others) 1990;49:410–2*

Surgical instruments

Can AIDS be prevented after injury with contaminated instruments? (Robicsek, Duncan, & others) 1990;49:984–6*

The Killingbeck pediatric lung retractor (Satur & Walker) 1990;49:489–90,HW

Surgical wound dehiscence

Recent experience with major sternal wound complications (Demmy, Park, & others) 1990;49:458–62*

Repair of sternal dehiscence using a Harrington compression system (Cochran & Rohrich) 1990;49:853,CP; Reply (Johnson) 1990;49:854,CP

Successful surgical management of bronchial dehiscence after single-lung transplantation (Kirk, Conacher, & others) 1990;49:147–9,CR

Survival

Cancer recurrence after resection: T1 N0 non-small cell lung cancer (Thomas, Rubinstein, & others) 1990;49:242–7*

Case-matched comparison of mitral valve replacement and repair (Craver, Cohen, & Weintraub) 1990;49:964–9*

Chemotherapy and radiation therapy before transhiatal esophagectomy for esophageal carcinoma (Orringer, Forastiere, & others) 1990;49:348–55*

Determinants of survival and valve failure after mitral valve replacement (Teoh, Ivanov, & others) 1990;49:643–8*

Esophagogastrectomy via left thoracophrenotomy (Page, Khalil, & others) 1990;49:763–6,CR

Heart valve operations in patients with active infective endocarditis (David, Bos, & others) 1990;49:701–5*

Life-threatening acute posterior mediastinitis due to esophageal perforation (Burnett, Rosemurgy, & Pfeiffer) 1990; 49:979–83*

Long-term results of direct aortoplasty for repair of aortic coarctation in adults (Heikkinen & Ala-Kulju) 1990;49: 948–50*

Lung and heart-lung transplantation (LeGal) 1990;49:840–4,CV

Operative treatment of *Fusarium* fungal infection of the lung (Lupinetti, Giller, & Trigg) 1990;49:991–2,CR

Predicted pulmonary function and survival after pneumonectomy for primary lung carcinoma (Putnam, Lammermeier, & others) 1990;49:909–15*

Surgery for high cervical esophageal carcinoma: experience with 11 patients (Mansour, Picone, & Coleman) 1990; 49:597–602*

Surgical excision of intracardiac myxomas: a 20-year follow-up (Bortolotti, Maraglino, & others) 1990;49:449–53*

Surgical treatment of cardiac tumors: a 25-year experience (Murphy, Sweeney, & others) 1990;49:612–8*

Survival after conservative resection for T1 N0 M0 non-small cell lung cancer (Read, Yoder, & Schaeffer) 1990;49: 391–400*

Survival after surgical resection for high-grade chest wall sarcomas (Perry, Venzon, & others) 1990;49:363–9*

Ten-year experience with porcine bioprosthetic valve: interrelationship of valve survival and patient survival in 1,050 valve replacements (Jones, Weintraub & others) 1990; 49:370–84*

Thoracic aortic aneurysms after acute type A aortic dissection: necessity for follow-up (Heinemann, Laas, & others) 1990; 49:580–4*

Swine

Atrial pressure and hormonal and renal responses to acute cardiac tamponade (Hynynen, Salmenpera, & others) 1990;49:632–7*

Swine (cont'd)
- Preliminary report of localization of spinal cord blood supply by hydrogen during aortic operations (Svensson, Patel, & others) 1990;49:528–36*
- Reduced-size lung transplantation in neonatal swine: technique and short-term physiological response (Crombleholme, Adzick, & others) 1990;49:55–60*

Tachycardia
- Computerized potential distribution mapping: a new intraoperative mapping technique for ventricular tachycardia surgery (Harada, Tweddell, & others) 1990;49:649–55*
- Skeletonization of the atrioventricular node for AV node reentrant tachycardia: experience with 32 patients (Guiraudon, Klein, & others) 1990;49:565–73*
- Surgical ablation for idiopathic ventricular tachycardia (Blakeman & Wilber) 1990;49:314–6,CR
- Surgical cure of automatic atrial tachycardia by partial left atrial isolation (Chang, Chang, & others) 1990;49:466–8,CR
- Surgical treatment of automatic atrial tachycardias (Hendry, Packer, & others) 1990;49:253–60*

Tetracycline
- Tetracycline pleurodesis for persistent air leak (Chechani) 1990;49:166–7,CP; Reply (Almassi & Haasler) 1990;49:167,CP

Tetralogy of Fallot
- C. Walton Lillehei and total correction of tetralogy of Fallot (Gott) 1990;49:328–32,CL
- Palliative reconstruction of right ventricular outflow tract in tetralogy with hypoplastic pulmonary arteries (Okita, Miki, & others) 1990;49:775–9*
- Use of bovine lung heparin to obviate anaphylactic shock caused by porcine gut hepatin (Harada, Tatsuno, & others) 1990;49:826–7,CR

Thoracic arteries
- Effect of omental, intercostal, and internal mammary artery pedicle wraps on bronchial healing (Turrentine, Kesler, & others) 1990;49:574–9*
- Results of internal thoracic artery grafting over 15 years: single versus double grafts (Fiore, Naunheim, & others) 1990;49:202–9*

Thoracic surgery
- Assessment of spinal cord blood supply during aortic operations (Blaisdell) 1990;49:522,ED
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- Direct access to the paravertebral space at thoracotomy (Berrisford & Sabanathan) 1990;49:855,CP
- Ectopic ossification after median sternotomy (Weiman) 1990;49:510,CP
- Esophagogastrectomy via left thoracophrenotomy (Page, Khalil, & others) 1990;49:763–6,CR
- Individual ligation technique for lower lobe lobectomy (Faber) 1990;49:1016–8,CL
- Long-term results of direct aortoplasty for repair of aortic coarctation in adults (Heikkinen & Ala-Kulju) 1990;49:948–50*
- Mediastinal epithelioid hemangioendothelioma (Toursarkissian, O'Connor, & Dillon) 1990;49:680–5,CV
- Operation for N2 small cell lung carcinoma (Salzer, Muller, & others) 1990;49:759–62*
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- Shone's anomaly: operative results and late outcome (Bolling, Iannettoni, & others) 1990;49:887–93*
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Thoracoscopy
- Thoracoscopic ablation of blebs (Chiu) 1990;49:851,CP; Reply (Wakabayashi) 1990;49:851,CP

Thoracotomy
- Direct access to the paravertebral space at thoracotomy (Berrisford & Sabanathan) 1990;49:855,CP

Thorax
- Can computed tomography of the chest stage lung cancer? Yes and no (Lewis, Pearlberg, & others) 1990;49:591–6*
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- Surgical techniques for chest wall reconstruction (Mathisen) 1990;49:164–5,KR
- Survival after surgical resection for high-grade chest wall sarcomas (Perry, Venzon, & others) 1990;49:363–9*

Thrombocytopenia
- Intraoperative management of patients with heparin-induced thrombocytopenia (Kappa, Fisher, & others) 1990;49:714–23*

Thromboembolism
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Herbert Sloan, MD

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1991 Annual Meeting of The Society of Thoracic Surgeons

The 1990 meeting is an Interim Meeting to be held at the Hyatt Regency Chicago in Chicago, Illinois, September 21–23, 1990. No abstracts are solicited for that program.

The Twenty-seventh Annual Meeting will be held at the San Francisco Moscone Center in San Francisco, California, February 18–20, 1991.

APPLICATION FOR MEMBERSHIP

(Deadline: September 1, 1990)

Requests for application forms for membership in The Society of Thoracic Surgeons should be addressed to James L. Cox, MD, Chairman, Membership Committee, The Society of Thoracic Surgeons, 111 E Wacker Dr, Chicago, IL 60601.

The completed application forms and supporting letters must be in the hands of the Membership Committee by **September 1, 1990,** for the applicant to be considered for election at the meeting in February 1991.

CALL FOR ABSTRACTS

(Deadline: September 12, 1990)

Abstracts for papers to be presented at the 1991 Annual Meeting are now being accepted. The deadline for receipt of abstracts is September 12, 1990. An original and one copy should be submitted, using the official Society abstract form (on verso of this page), to Alexander S. Geha, MD, and Robert A. Guyton, MD, Co-Chairmen, Program Committee (Abstracts), The Society of Thoracic Surgeons, 111 E Wacker Dr, Chicago, IL 60601. Additional forms are available upon request from The Society. Abstracts must summarize an original contribution not presented or accepted for presentation elsewhere. Abstracts must be in the hands of the Program Committee by **September 12, 1990.** Abstracts received after the deadline will not be considered. It is requested that the covering letter indicate the author who is to receive notice of acceptance or rejection and the author who will present the paper.

The Thoracic Surgery Directors Association (TSDA) has established a Resident Presentation Award for 1991. Potential TSDA Resident Award abstracts must represent original work by the resident, who is to be first author and presenter. TSDA Resident Award abstracts may be directed to the regular Scientific Session or the Research Forum.

Essayists are reminded that according to the bylaws of The Society, all papers accepted for presentation: (1) must be submitted in *triplicate* to the Secretary at the time of presentation or to the Editor of *The Annals of Thoracic Surgery* prior to presentation; and (2) become the property of The Society if they are accepted for presentation.

It is well recognized that abstracts frequently belie their content by virtue of poor organization, and as a result valuable contributions to the Scientific Sessions may be unrecognized. It is important that methods and results be clearly defined in the abstract; therefore, general statements such as "results will be presented" are not acceptable. When a procedure or method is stated to have "advantages," those advantages should be defined. Abstracts that simply summarize results without defining objectives and methods are equally undesirable.

Speakers for accepted papers are limited to ten minutes.

Instructions for Abstracts

The official abstract form printed on verso of this page and available from The Society, or photocopies thereof, must be used. **Authors must categorize the principal subject of their abstract by checking the appropriate box.**

If the author wishes to submit the abstract for consideration for the Thoracic Surgery Directors Association Award competition, the *additional* appropriate box must be checked. Also, the name of the Program Director must appear on a TSDA Resident Award abstract.

1. Full names of authors must appear on the abstract.
2. Institution or address is to be given, but omit titles, degrees, and appointments.
3. Abstracts must be limited to 250 words and must be typed double-spaced.
4. When percentages are used, the absolute numbers of derivation must be stated.
5. The body of the abstract should include:
    a. A brief statement of the purpose of the study being presented, with reference to the current state of research in the field.
    b. The methods of study or experimental approach clearly, but briefly, defined.
    c. A summary of the results and/or conclusions of the study, including sufficient details to support those conclusions.
    d. A statement concerning the significance of the work and its possible implications for further research.
6. The form must be signed by the senior author attesting that the material will not be presented elsewhere if accepted for the scientific program nor published elsewhere if accepted for publication in *The Annals of Thoracic Surgery.*

In discussion of papers, The Society encourages a healthy spirit of constructive critical review and rebuttal by comments pertinent to the content of the paper being presented. Presentation of discussion in the form of "secondary papers" will not be welcomed.

CALL FOR SURGICAL FILMS OR VIDEO TAPES

(Deadline: September 1, 1990)

The popular surgical movie program will again be presented at the San Francisco meeting. The Program Committee hereby solicits the submission of surgical films, which may portray a surgical procedure or simply a technical maneuver that the surgeon thinks will be of interest to The Society. The film may not exceed ten minutes in length. Sound movies are acceptable but not necessary; however, live narration by the principal surgeon is a requirement for participation in the movie night program. The movies should be on 16-mm film or ¾-inch U-Matic video tape. The deadline for submission is **September 1, 1990.** Films and video tapes also should be sent to Alexander S. Geha, MD, and Robert A. Guyton, MD, Co-Chairmen, Program Committee, at The Society of Thoracic Surgeons, 111 E Wacker Dr, Chicago, IL 60601.

Richard P. Anderson, MD
Secretary

For Committee use only:

THE SOCIETY OF THORACIC SURGEONS

Abstract Form

For Committee use only:
Rating: (Highest 1)

1☐ 2☐ 3☐ 4☐ 5☐

LIMIT TO 250 WORDS AND DOUBLE-SPACE

TITLE

AUTHOR(S)

ADDRESS

PLEASE CHECK ONE APPROPRIATE CATEGORY

- ☐ **Pulmonary**
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- ☐ **Chest wall Mediastinum**
- ☐ **Trauma**
- ☐ **Transplantation**
- ☐ **Heart**
- ☐ **Great vessels**
- ☐ **Valves**
- ☐ **Congenital**
- ☐ **Coronary**
- ☐ **Electrophysiology**
- ☐ **Myocardial protection**
- ☐ **Extracorporeal circulation**
- ☐ **Endoscopy**
- ☐ **Vascular surgery**
- ☐ **Research, Cardiac**
- ☐ **Research, Congenital Cardiac**
- ☐ **Research, Transplantation**
- ☐ **Research, General Thoracic**

☐ **PLEASE CHECK IF ALSO SUBMITTED FOR THE THORACIC SURGERY DIRECTORS ASSOCIATION AWARD**

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I acknowledge that the material will not be presented elsewhere if accepted for presentation on The Society of Thoracic Surgeons Scientific Program. I further understand that the Editorial Board of *The Annals of Thoracic Surgery* has the right of first refusal for publication of this material.

____

Signature of Senior Author

Information About Membership in The Society of Thoracic Surgeons

The Society of Thoracic Surgeons (STS) is the largest organization of its kind devoted primarily to the practice of thoracic and cardiovascular surgery. The major purpose of The Society is educational, with the aim of providing thoracic and cardiovascular surgeons from both academic and private practice environments a forum for presentation and discussion of scientific topics. As outlined in the original bylaws of The Society, this educational emphasis is designed to accomplish the broader goal of improving the quality and practice of thoracic and cardiovascular surgery as a specialty.

The STS recently celebrated its 25th Anniversary, having been founded in 1964. Since its inception, The Society has grown continually so that at present the membership includes the majority of board certified thoracic surgeons in the United States as well as thoracic and cardiovascular surgeons from 45 other countries.

In addition to an annual scientific meeting, the STS has more than 20 committees working on diverse matters related to improving thoracic and cardiovascular surgery as a specialty. Examples of these committees are: Standards and Ethics, Government Relations, Medico-Legal Affairs, Surgical Standby for PTCA, Physician Reimbursement, CPT-4 Advisory, Education and Resources, and Cardiothoracic Surgical Practice Guidelines. The Committee to Analyze Circulatory Assist Devices and Artificial Hearts sponsors topical meetings (next meeting, November 15–17, 1991, in San Francisco). The Committee to Develop a National Database for Thoracic Surgery plans to implement the first comprehensive database program for thoracic and cardiovascular surgeons in the United States; it will be offered to members in the near future.

There are five types of membership in The Society of Thoracic Surgeons: active, senior, honorary, international, and candidate.

Active Member Applicants for active membership must be surgeons whose practice is devoted primarily to the field of thoracic and cardiovascular surgery.

QUALIFICATIONS: Applicants must be Diplomates of the American Board of Thoracic Surgery or Fellows in The Royal College of Surgeons of Canada, and be established in the practice of thoracic and/or cardiovascular surgery for a minimum of three years. There must be sponsoring letters from three active STS members.

Senior Member Any active member or international member, upon reaching the age of sixty-five years, shall automatically become a Senior Member.

Honorary Member Honorary Membership shall be reserved for distinguished persons worldwide who have achieved prominence in the field of thoracic and cardiovascular surgery and have contributed to its advancement. Honorary members shall be elected by a majority vote of The Society at its annual business session upon recommendation of the Council of the STS.

International Member Members from countries other than the United States and Canada were previously accepted only as active members. Because of differences in training and practice, the application process for foreign members was often complex and time consuming. To allow for greater participation by thoracic and cardiovascular surgeons from other countries, the bylaws have been revised to provide for a category of International Member, which is the category for all members outside the United States and Canada.

QUALIFICATIONS: Applicants shall reside outside the United States and Canada. Their training in this specialty shall be considered equivalent to that of Active Members. They shall have achieved appropriate certification or recognition of achievements in this specialty in their country and must devote the majority of their professional efforts to the interests of the specialty. Appropriate documentation of professional achievements shall be provided to the Membership Committee.

Candidate Member Candidate membership was created to allow participation of younger surgeons in the time prior to being qualified for active membership.

QUALIFICATIONS: Applicants shall be enrolled in a thoracic and cardiovascular training program accredited by the Residency Review Committee for Thoracic Surgery under the authority of the Accreditation Council for Graduate Medical Education or in a similar program approved for training by The Royal College of Surgeons of Canada. Also, individuals who have completed their training in one of the above programs and are in process of acquiring certification by the American Board of Thoracic Surgery or The Royal College of Surgeons of Canada are eligible for this type of membership.

If you would like to receive an application for membership in The Society, send the request printed below. This year's deadline for receipt of applications is September 1, 1990. We suggest you apply soon as it takes time to process the application. Approved applications will be presented to the membership at the February 1991 Annual Meeting in San Francisco.

REQUEST FOR APPLICATIONS

Please send an application for: ☐ Active membership ☐ International membership ☐ Candidate membership

TO: NAME: ____________________

ADDRESS: ____________________

____

TELEPHONE NUMBER: ____________________

Send to: James L. Cox, MD, Chairman, Membership Committee
The Society of Thoracic Surgeons, 111 East Wacker Drive, Suite 600, Chicago, IL 60601
Telephone: (312) 644-6610; FAX: (312) 938-1214

THE ANNALS OF THORACIC SURGERY
Official Journal of The Society of Thoracic Surgeons and the Southern Thoracic Surgical Association

EVENTS OF INTEREST

Brief notices of meetings, courses, and symposia relevant to cardiothoracic surgery are published as a service. They should be formatted as follows: meeting title, date, place, sponsor, accreditation, director, fee, minimum/maximum attendance, and address/phone for further information. Announcements should be sent at least 4 months in advance of publication to *The Annals of Thoracic Surgery*, 3108 Queeny Tower, Barnes Hospital Plaza, St. Louis, MO 63110-1041; telephone, (314) 361-6084.

Internal Thoracic Artery for Myocardial Revascularization, Toulouse, France—June 27–29, 1990

For information on this meeting, contact the Secretary of the Symposium, Department of Cardiovascular Surgery, Rangueil University Hospital, 31054 Toulouse Cedex, France; or telephone 61.32.26.51 or 61.32.26.53 (telex: 530 826 F).

Cardiac Arrhythmia Surgery International Seminar I, Geneva, Switzerland—June 27–29, 1990

For information on this meeting, contact Beverly Farrier, ProMedica International, 620 Newport Center Drive, Suite 575, Newport Beach, CA 92660; or telephone (714) 640-5870 (facsimile: (714) 759-6911).

New Devices for the Cardiac and Peripheral Vascular Laboratories, Orlando, Florida—July 2–6, 1990

This meeting is sponsored by the Pittsburgh Vascular Institute and the Pittsburgh Heart Institute of Shadyside Hospital, Pittsburgh, PA, and is accredited for 15 hours in category 1 of the Physician's Recognition Award of the AMA. For information on this meeting, contact Shadyside Hospital, New Devices Technology Workshop, 5230 Centre Ave, Pittsburgh, PA 15232; or telephone (412) 622-1051.

Seventh Congress of the European Society of Biomechanics, Aarhus, Denmark—July 8–11, 1990

For information on this meeting, contact Seventh Congress of ESB, Biomechanics Laboratory, Orthopaedic Hospital, Randersvej 1, 8200 Aarhus N, Denmark; or telephone 45-616-7500, extension 4649.

Myocardial Preservation Into the 21st Century, Oxford, England—August 12–15, 1990

For information on this meeting, contact Beverly Farrier, ProMedica International, 620 Newport Center Drive, Suite 575, Newport Beach, CA 92660; or telephone (714) 640-5870 (facsimile: (714) 759-6911).

Thirty-ninth Annual Meeting of the Scandanavian Association for Thoracic and Cardiovascular Surgery and the Scandanavian Society for Extracorporeal Technology, Reykjavík, Iceland—August 29–31, 1990

For information on this meeting, contact G. Ólafsson, MD, Chairman, Department of Thoracic and Cardiovascular Surgery, Landspítalinn, University Hospital, Reykjavík, Iceland.

Thirty-ninth Congress of the European Society for Cardiovascular Surgery, Budapest, Hungary—September 9–12, 1990

For information on this meeting, contact Budapest Convention Centre, H-1444, PO Box 233, Budapest XI, Jagello u 1-3, Hungary; or telephone (361) 852-507 (facsimile: (361) 665-636; telex: 22-7717).

Fourth Annual Meeting of the European Association for Cardio-thoracic Surgery, Naples, Italy—September 17–19, 1990

For information on this meeting, contact the Organizing Secretariat, MCM, Rione Sirignano, 5, 80121 Naples, Italy; or telephone (0)81/7611085-7611277 (facsimile: (0)81/664372; telex: 710626).

Second Annual Meeting of the International Society for Applied Cardiovascular Biology, Venice Lido, Italy—September 17–20, 1990

For information on this meeting, contact the Congress Secretariat, Kele Teo, San Marco 4930, Venice, Italy.

Interim Meeting of The Society of Thoracic Surgeons, Chicago, Illinois—September 22–23, 1990

For information on this meeting, contact Richard P. Anderson, MD, The Society of Thoracic Surgeons, 111 East Wacker Dr, Chicago, IL 60601-4301; or telephone (312) 644-6610.

Mechanical Circulatory Support 1990, London, England—September 26–28, 1990

For information on this meeting, contact Anne Sumner, Mechanical Circulatory Support 1990, Medical Research Services, 60 Oxford St, Southampton SOI 1DL, Hants, England; or telephone 0703 231068.

Controversies in Cardiovascular Disease: Surgical Versus Medical Treatment, Aalst, Belgium—September 28–29, 1990

For information on this meeting, contact Dr F. Wellens, Department of Cardiovascular Surgery, O.L. Vrouwziekenhuis, Moorselbaan 164, B-9300 Aalst, Belgium; or telephone 053/78 53 53 (facsimile: 053/78 38 76).

Thirty-seventh Annual Meeting of the Southern Thoracic Surgical Association, Cerromar Beach, Puerto Rico—November 8–10, 1990

For information on this meeting, contact Gordon F. Murray, MD, Southern Thoracic Surgical Association, 111 East Wacker Dr, Chicago, IL 60601-4301; or telephone (312) 644-6610 (facsimile: (312) 938-1214).

CLASSIFIED ADS

Classified ads for positions wanted or available must relate only to the thoracic or cardiovascular surgical specialties. Display space (minimum, ¼ page) is also available through Media for Medicine, Inc, 440 Park Avenue South, New York, NY 10016 (telephone: (212) 684-5540; facsimile: (212) 545-0651). All ads are subject to the approval of the editor, and material deemed inappropriate for publication in *The Annals* will be rejected. If anonymity is desired, please indicate this at the time of submission and a code number will be assigned. All responses received will remain confidential in the publisher's office and will be conveyed to the advertiser shortly after receipt. The charge for all classified advertising is **$1.10 per word per insertion, minimum 20 words.** Abbreviations, dates, initials, post office box numbers, telephone numbers, years, and zip codes are considered one word each. **There is an additional fee of $15.00 per insertion for box number ads.** The copy deadline is 7 weeks prior to publication, eg, for the March issue, copy should be received by the 1st of January. Ad orders should be for a maximum of 6 months; ads may be renewed for up to 6 months at a time. Ad copy should be typed double-spaced and mailed in duplicate to: *The Annals of Thoracic Surgery*, Desk Editorial, Classified Ads, Elsevier Science Publishing Co, Inc, 655 Avenue of the Americas, New York, NY 10010 (facsimile: (212) 633-3695). Make *nonrefundable* check payable to Elsevier Science Publishing Co, Inc.

Please note that *The Annals* does not function as a placement service and cannot provide information on advertised positions. When answering advertisements, please write individually to each advertiser you wish to contact, using the address given in the ad. To respond to an anonymous ad, write to Box Number, *The Annals of Thoracic Surgery*, Elsevier Science Publishing, 655 Avenue of the Americas, New York, NY 10010.

SITUATIONS AVAILABLE

Cardiothoracic surgeon wanted for a busy, expanding, 3-man, private practice group in the Midwest. Applicant must be ABTS eligible or certified. Prefer recent graduate with some background in electrophysiologic surgery. Please send curriculum vitae.

Please respond to Box 189. 189H/G

Associate Director for Heart Transplantation is urgently needed for rapidly expanding heart transplantation program in California.

Please respond with curriculum vitae to the Pacific Cardiothoracic Surgery Group, 201 S Alvarado St, Suite 626, Los Angeles, CA 90057. 240L/K

Cardiovascular and thoracic surgeon, board eligible/certified, wanted to join well-established private practice located in Ft. Lauderdale, FL. Send CV.

Please respond to Box 251. 251L/K

Cardiovascular and thoracic surgeon, board eligible or certified, wanted to join another surgeon in private practice in the Southeast. Please respond with curriculum vitae.

Please respond to Box 252. 252L/F

Cardiovascular/thoracic surgeon to complete 3-man group in adult cardiothoracic private practice in Michigan. Must be board eligible or certified. References required.

Please respond to Box 269. 269A/F

Cardiothoracic surgeon, board certified with 2 to 4 years experience in cardiac surgery, wanted to join a growing group of cardiac surgeons, cardiologists, and pulmonologists in the Pacific Northwest. General thoracic and vascular experience necessary. Research opportunities available, academic links possible. Salary leading to full partnership in 2 or 3 years; potential exists for leadership in major cardiac program.

Please send curriculum vitae to John R. Bauman, Administrator at The Thoracic Clinic, PC, 507 NE 47th Ave, Portland OR 97213-2282. 274A/F

Cardiovascular and thoracic surgeon: Position available for board certified or board eligible person to join busy practice involved in adult cardiac, thoracic, and vascular surgery in major New England city. Association will lead to partnership. Excellent fringe benefits. Please send curriculum vitae.

Please respond to Box 291. 291B/G

Thoracic/vascular surgeon to join small group private practice in medium-sized New England community. BC/BE in thoracic surgery. Prefer C/E for Cert. Special Competence in Vascular Surgery. Recent medical graduate preferred. University-affiliated hospital. Full partnership in 3 years.

Please respond to Box 301. 301C/F

Cardiothoracic/vascular surgeon wanted to join established practice in Ohio. Recent graduate preferred. Please submit curriculum vitae and three references.

Please respond to Box 302. 302C/I

Group of two cardiothoracic surgeons seeking third associate. Recent graduate preferred. American Board of Thoracic Surgery certification or eligibility is required. Practice consists of adult cardiac surgery at University-affiliated hospital in the New York metropolitan area.

Please respond to Box 310. 310D/F

Cardiovascular surgeon to become second surgeon in new (two year old) adult cardiac program on west coast of Florida.

Submit CV to Manatee Cardiac Surgeons, PA, 201 4th Ave East #2, Bradenton, FL 34208. 312D/F

Cardiac surgeon wanted to join established private practice in Eastern PA. Primarily adult cardiac. BC/BE required. Please send CV.

Please respond to Box 315. 315D/I

Cardiovascular and thoracic surgeon, board eligible or certified, wanted to join another physician in private practice in Northeast. Immediate availability. Please respond with curriculum vitae.

Please respond to Box 316. 316D/I

Cardiac surgeon with additional interest and experience in vascular surgery wanted to join large, active group practice in suburban Washington, DC. Prefer recent ABTS-certified surgeon with 2 to 5 years experience.

Please respond to Box 322. 322D/F

Cardiovascular thoracic surgeon—board certified/board eligible and recently trained to join an established, busy, dynamic, and expanding private practice in northwest Indiana. The sphere of practice is approximately 30 minutes from downtown Chicago. Guaranteed excellent starting salary with progressive future growth and comprehensive corporate fringe benefit program.

Write including CV and details of experience and capabilities to PO Box 3066, Munster, IN 46321. 329E/F

Excellent opportunity for recent graduate, board eligible or certified, for private practice of cardiac, thoracic, and major vascular cases in Great Lakes area. Include references and curriculum vitae.

Please respond to Box 335. 335E/G

Cardiothoracic surgeon needed for expanding program in mid-Michigan. Primarily adult cardiac and thoracic. BC/BE required. Please send CV with references.

Please respond to Box 337. 337E/J

Cardiovascular and thoracic surgeon: Position available for a board certified or eligible cardiovascular and thoracic surgeon to join a 2-man private practice involved with adult cardiac, thoracic, and peripheral vascular surgery in a desirable eastern metropolitan area. Association features competitive salary, excellent fringe benefits, and a goal of early partnership. Please send curriculum vitae.

Please respond to Box 339. 339E/G

Cardiothoracic surgeon: Excellent opportunity in mid-West for experienced cardiothoracic surgeon to join highly regarded board-certified physician who is Chief of Cardiothoracic Service at 450-bed community teaching hospital. Very active practice in adult open hearts and general thoracic. Private practice association leading to partnership.

Please contact Jeanne Dowrick, Fulton, Longshore and Associates, 527 Plymouth Rd, Plymouth Meeting, PA 19462; telephone (800) 346-8397 or (215) 834-6780. 341E/G

Cardiothoracic surgeon wanted for busy, expanding Southeastern private group in regional medical center with university affiliation. Practice includes most aspects of adult and pediatric cardiac and thoracic surgery. Surgeon must be board eligible or certified with contemporary training. Excellent opportunity for suitable applicant.

Please respond to Box 342. 342F/I

Thoracic-cardiovascular surgeon to develop the cardiovascular program in an acute care facility located in the South. This open-heart program will be the prime program in an 18-county serving area. Association with a well-established 12-physician group.

Please respond to Box 344. 344F

Thoracic cardiovascular surgeon to join group of 12 surgeons located in the South. Group also includes physician assistants, perfusionists, perfusion assistants, surgical technicians, registered nurses, and ancillary office personnel. Group philosophy is to practice quality medicine with utmost concern for patient.

Please respond to Box 345. 345F

Cardiothoracic surgeon: The Oregon Health Sciences University, Division of Cardiopulmonary Surgery, with strong active programs in all aspects of cardiopulmonary surgery and heart transplantation, has an academic position available at the Assistant Professor level. Candidates should be eligible for or certified by the American Board of Thoracic Surgery. Outstanding opportunity for an individual with excellent clinical skills, a track record of academic productivity, and a commitment to research and teaching. Two to four years of post-residency clinical experience is highly desirable.

Applicants are invited to respond with curriculum vitae to Adnan Cobanoglu, MD, Professor and Chief, Division of Cardiopulmonary Surgery, The Oregon Health Sciences University, 3181 SW Sam Jackson Park Rd, Portland, OR 97201. 346F/G

Thoracic surgery residency: Residency in thoracic surgery. Position available July 1991 at the University of Massachusetts Medical School/Hospital. Three-year program includes training in adult and pediatric cardiac and general thoracic surgery.

Interested candidates may contact Thomas J. Vander Salm, MD, Department of Surgery, University of Massachusetts Medical Center, 55 Lake Ave North, Worcester, MA 01655. 347F/H

Cardiovascular and thoracic surgeon: Excellent opportunity to join a busy north central Florida group. Practice specializes in adult cardiac and thoracic surgery. Excellent benefit package. Please reply with curriculum vitae.

Please respond to Box 348. 348F/K

Cardiovascular and thoracic surgeon, BE/BC, to join in 2-man private practice in cardiovascular and thoracic surgery in the Northeast. Excellent benefits package. Association will lead to partnership. Please send curriculum vitae.

Please reply to Box 350. 350F/K

SITUATIONS WANTED

Cardiothoracic surgeon, 34, American, NE university training, 10 months private practice experience, wishes to relocate. ABS, ABTS eligible. Experienced in adult coronary reoperations, ventricular assists, and electrophysiologic surgery. Seeks practice in Southwest/Southeast. Curriculum vitae and references upon request.

Please respond to Box 318. 318D/G

Cardiovascular and thoracic surgeon, 41, University trained, wishes to relocate. ABS and ABTS certified, member of the STS, ICS, ISHT, ACC, and AHA. Author of several publications. Currently experienced in IMA surgery, valve repair and replacement. Ten years ago developed a successful and busy pediatric cardiac surgery program, with extensive experience in neonates and infants.

Please respond to Box 343. 343F/H

FELLOWSHIPS

Clinical fellowship in adult cardiothoracic surgery available July 1, 1990. Fellowship experience will include acquired cardiac, general thoracic, esophageal, and pulmonary surgery and diagnostic and laser therapeutic endoscopic procedures. Competitive salary and benefits package offered. Minimum requirement: American Board of Surgery certification or eligibility.

Please send resume to Division of Cardiothoracic Surgery, University of Missouri-Columbia, School of Medicine, One Hospital Drive, Columbia, MO 65212. 332E/F

Fellowship in thoracic and cardiovascular surgery available July 1, 1990. Good salary and all expenses paid. Research opportunities.

Please send inquiries to George C. Morris, Jr, MD, and Gerald M. Lawrie, MD, Baylor College of Medicine, Houston, TX 77030; (713) 797-9428. 349F

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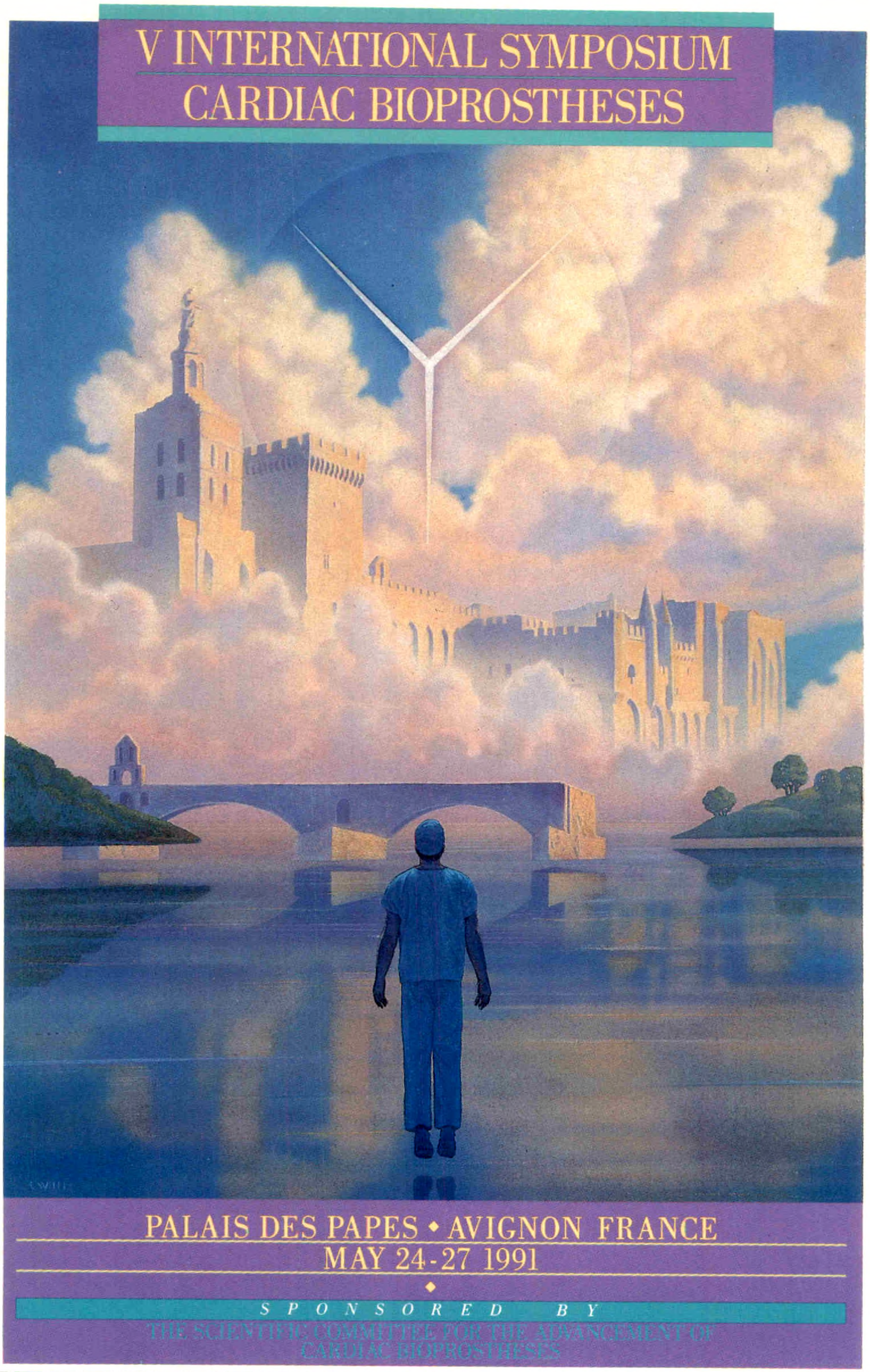

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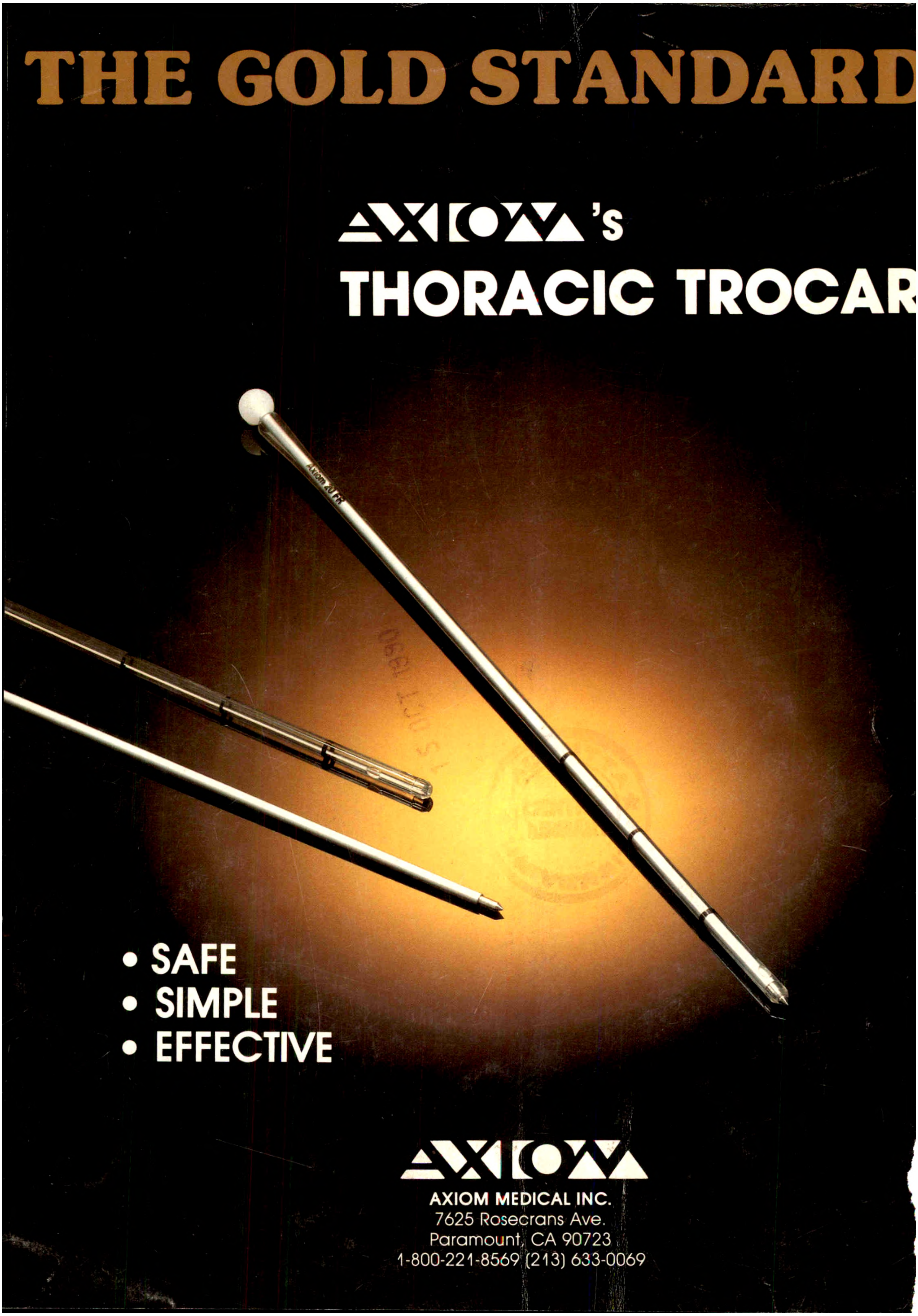

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